GSEELRSLYNTVATLYCVHQ

QUERY	GSEELRSLYNTVATLYCVHQ
CONSENSUS_A	-T------------------
A.KE.Q23-CXC-CG	-T--IK--F-----------
A.SE.SE6594	-T--IK--F-----------
A.SE.SE7253	-T------F----V------
A.SE.SE7535	-T------------------
A.SE.SE8131	-T------------------
A.SE.SE8538	-T---K--------W----
A.SE.SE8891	-T------------------
A.UG.92UG037	-T------------------
A.UG.U455	-T-----------V------
CONSENSUS_B	--------------------
B.AU.AF128998	-----K----A---------
B.-.NL43E9	-----------I-A------
B.AU.MBC18	-----K-V--A--V------
B.AU.MBC200	----IK--------------
B.AU.MBC925	---DF---------------
B.AU.MBCC54	--------------------
B.AU.MBCC98	---D---V------------
B.AU.MBCD36	-----K-------V------
B.CN.RL42	--------F----------L
B.DE.D31	--------F-----------
B.DE.HAN	--------------------
B.ES.89SP061	--------------------
B.FR.HXB2	--------------------
B.GA.OYI	----I---------------
B.GB.CAM1	--------------------
B.GB.MANC	-----K-------V------
B.JP.JH31	-----K--F-----------
B.NL.3202A21	--------F----V------
B.TW.LM49	-----------------I--
B.US.85WCIPR54	--------H----V------
B.US.AD8	-----K--F-----------
B.US.BC	-----K-----I-V------
B.US.DH123	-------------------E
B.US.JRCSF	-----T--------------
B.US.JRFL	--------------------
B.US.MNCG	-----K--------------
B.US.NC7	-----------I--------
B.US.NY5CG	----R---F----V------
B.US.P896	-----K--------------
B.US.RF	-----K----A---------
B.US.SF2	--------------------
B.US.WC001	--------H----V------
B.US.WEAU160	-------------V------
B.US.WR27	--------F-----------
B.US.YU2	--------------------
CONSENSUS_C	-T------?----------?
C.BR.92BR025	-TK--I--H----------E
C.BW.96BW01B22	-T---K------------E
C.BW.96BW0402	-T------F----------K
C.BW.96BW0502	-T----------------A
C.BW.96BW1104	-T--I-------------E
C.BW.96BW1210	-T---K------------E
C.BW.96BW15B03	-T------F----------E
C.BW.96BW1626	-T---K-------V-F---A
C.BW.96BW17A09	-T---K--------------
C.ET.ETH2220	-T---K--F-----------
C.IN.93IN904	-T------H---V------A
C.IN.93IN905	-T------F----------A
C.IN.93IN999	-T------H----------E
C.IN.94IN11246	-T------F----------A
C.IN.95IN21068	-T------F----------A
CONSENSUS_D	--------------------e
D.CD.84ZR085	-------I-----------K
D.CD.ELI	-T-----------------K
D.CD.NDK	----I----------E
D.CD.Z2Z6	--------F----------E
D.UG.94UG1141	----IK------V------E
CONSENSUS_F	-------------V--f---
F.BR.BZ162	-------------V--F---
F.CD.VI174	--------F--IVV--Y---
F.RW.VI69	-------------V--F---
CONSENSUS_F1	-----?--?--?-V--y---
F1.BE.VI850	-----K--F----V--Y---
F1.BR.93BR020.1	-----K-----I-V--Y---
F1.FI.FIN9363	-----------I-V--F---
F1.FR.MP411	--------F----V------
CONSENSUS_F2	-----K--?-??VV--Y---
F2.CM.MP255	-----K----A-VV--Y---
F2.CM.MP257	-----K--F--IVV--Y---
CONSENSUS_G	-T--IK--F-----------
G.BE.DRCBL	-T--IK--F-----------
G.FI.HH8793	-T--IK--F-----------
G.NG.92NG083	-T------F-----------
G.SE.SE6165	-T--IK----A---------
CONSENSUS_H	-T---Q--F----V------
H.BE.VI991	-T-D-Q-----I-V------
H.BE.VI997	-T---x--F---------L-
H.CF.90CF056	-T---K--F-L--V-----R
CONSENSUS_J	-T?-IK--------------
J.SE.SE9173	-T--IK--------------
J.SE.SE9280	-TQ-IK--------------
CONSENSUS_K	-----?--?----?------
K.BE.VI325	-----K--F---V-------
K.CD.EQTB11C	--------F-------W---
K.CM.MP535	----IK-----I-V--F---
N.CM.YBF30	----------AL-V-----S
CONSENSUS_O	--??-?--W-AI?V-W---N
O.CM.ANT70C	--DS-Q--W-AIVV-W---N
O.CM.MVP5180	---D-K--W-AI-V-W---N
CRF01-AE.CF.90CF40	-----K--F--I---W----
CRF01-AE.TH.93TH25	-----K-----I---W----
CRF01-AE.TH.CM240	-L---K--F------W----
CRF01-AE.TH.TH022	--------F------W----
CRF01-AE.TH.TH047	--------F--IV--W----
CRF02_AG.FR.DJ263	-----K-----I---W---K
CRF02_AG.FR.DJ264	-----K-----I---W----
CRF02_AG.NG.IBNG	-----K--F--I---W----
CRF03_AB.RU.KAL15	-----K--------------
CRF04_cpx.CY.94CY0	-----------IT--W----
CRF04_cpx.GR.97PVC	----VK--F--L---W----
CRF04_cpx.GR.97PVM	-----K--F-LI---W----
AC.ET.E3099G	-----K--------------
AC.IN.21301	-T------H----------A
AC.RW.92RW009	-TD-----------------
AC.SE.SE9488	-T--IK--F-----------
AC.ZM.ZAM174-21	-T------F--A-------E
AC.ZM.ZAM184	-T-DI--------V--Y---
AC.ZM.ZAM716-17	-T------F----------A
ACD.SE.SE8603	-T-------------W---K
AD.SE.SE6954	-----K--F----------A
AD.SE.SE7108	-T---K--------------
ADHU.NO.NOGIL3	-----K--F-L--V-W----
ADU.CD.MAL	----IK--------------
AG.NG.G3	-T--IK--F-----------
AG.SE.SE7812	-----K--F--I---W----
AGHU.GA.VI354	-----K--F-----------
AGJ.AU.BFP90	-----K--F-----------
AGJ.ML.95ML8	-----K--------------
AGU.CD.Z321	-T--II--------------
BF.BR.93BR029.4	--------------------
DF.CD.VI961	-------------------E
U.CD.VI1126	--------F---V---W---
CONSENSUS_CPZ	---g----F--l-V-W---s
CPZ.CD.CPZANT	R-P-II--F--ICV-W---K
CPZ.GA.CPZGAB	---G----F--L-V-W-I-S
CPZ.US.CPZUS	---G----F--L-V-W---S

MFSALSEGATPQDLNTMLNT

QUERY	MFSALSEGATPQDLNTMLNT
CONSENSUS_A	----------m---i
A.KE.Q23-CXC-CG	----------M---I
A.SE.SE6594	----------M---I
A.SE.SE7253	V---------M---I
A.SE.SE7535	----------M---I
A.SE.SE8131	----------H---M---I
A.SE.SE8538	----------I
A.SE.SE8891	----------G---M---I
A.UG.92UG037	----------M---I
A.UG.U455	----------M---V
CONSENSUS_B	----------
B.AU.AF128998	----------
B.-.NL43E9	----------
B.AU.MBC18	----------
B.AU.MBC200	----------
B.AU.MBC925	----------
B.AU.MBCC54	----------
B.AU.MBCC98	----------
B.AU.MBCD36	--T---------
B.CN.RL42	----------
B.DE.D31	----------
B.DE.HAN	----------
B.ES.89SP061	----------
B.FR.HXB2	----------
B.GA.OYI	-----A----------
B.GB.CAM1	----------
B.GB.MANC	---------I----------
B.JP.JH31	----------
B.NL.3202A21	----------
B.TW.LM49	----------
B.US.85WCIPR54	----------
B.US.AD8	----------
B.US.BC	----------
B.US.DH123	----------
B.US.JRCSF	----------
B.US.JRFL	----------
B.US.MNCG	----------
B.US.NC7	----------
B.US.NY5CG	----------
B.US.P896	----------
B.US.RF	----------
B.US.SF2	----------
B.US.WC001	----------
B.US.WEAU160	----------
B.US.WR27	----------Y-----
B.US.YU2	----------
CONSENSUS_C	--T---------
C.BR.92BR025	--T---------
C.BW.96BW01B22	--T---------
C.BW.96BW0402	--T---------
C.BW.96BW0502	--T---------
C.BW.96BW1104	--T----------T-
C.BW.96BW1210	--T---------
C.BW.96BW15B03	--T---------
C.BW.96BW1626	--T---------
C.BW.96BW17A09	--T---------
C.ET.ETH2220	--T---------
C.IN.93IN904	--T---------
C.IN.93IN905	--T---------
C.IN.93IN999	--T---------
C.IN.94IN11246	--T---------
C.IN.95IN21068	--T---------
CONSENSUS_D	----------
D.CD.84ZR085	----------
D.CD.ELI	----------
D.CD.NDK	----------
D.CD.Z2Z6	----------
D.UG.94UG1141	----------
CONSENSUS_F	----------
F.BR.BZ162	----------
F.CD.VI174	----------
F.RW.VI69	----------
CONSENSUS_F1	----------
F1.BE.VI850	----------T--------
F1.BR.93BR020.1	----------
F1.FI.FIN9363	----------
F1.FR.MP411	----------
CONSENSUS_F2	----------
F2.CM.MP255	----------
F2.CM.MP257	----------
CONSENSUS_G	----------xx-
G.BE.DRCBL	--T---------
G.FI.HH8793	----------
G.NG.92NG083	----------
G.SE.SE6165	----------L----
CONSENSUS_H	----------A----
H.BE.VI991	----------A----
H.BE.VI997	----------A----
H.CF.90CF056	----------A----
CONSENSUS_J	----------
J.SE.SE9173	----------
J.SE.SE9280	----------
CONSENSUS_K	----------
K.BE.VI325	-----AD----------
K.CD.EQTB11C	----------
K.CM.MP535	--T---------
N.CM.YBF30	--M--------S--------
CONSENSUS_O	--M------??Y-I-----A
O.CM.ANT70C	--M------ISY-I-----A
O.CM.MVP5180	--M------V-Y-I-----A
CRF01-AE.CF.90CF40	----------M---I
CRF01-AE.TH.93TH25	----------M---I
CRF01-AE.TH.CM240	----------M---I
CRF01-AE.TH.TH022	----------M---I
CRF01-AE.TH.TH047	----------M---I
CRF02_AG.FR.DJ263	--T----------M---I
CRF02_AG.FR.DJ264	--T----------M---I
CRF02_AG.NG.IBNG	----------M---I
CRF03_AB.RU.KAL15	----------M---I
CRF04_cpx.CY.94CY0	----------M---I
CRF04_cpx.GR.97PVC	----------M---I
CRF04_cpx.GR.97PVM	----------M---I
AC.ET.E3099G	----------
AC.IN.21301	--T---------
AC.RW.92RW009	--T---------
AC.SE.SE9488	--T---------
AC.ZM.ZAM174-21	--T---------
AC.ZM.ZAM184	----------
AC.ZM.ZAM716-17	--T---------
ACD.SE.SE8603	----------M---I
AD.SE.SE6954	---------A--------S-
AD.SE.SE7108	----------M---I
ADHU.NO.NOGIL3	------D--------M---I
ADU.CD.MAL	----------M---I
AG.NG.G3	--T---------
AG.SE.SE7812	----------M---I
AGHU.GA.VI354	----------M---I
AGJ.AU.BFP90	--T----------M---I
AGJ.ML.95ML8	----------M---I
AGU.CD.Z321	----------
BF.BR.93BR029.4	----------
DF.CD.VI961	--T---------
U.CD.VI1126	--T---------
CONSENSUS_CPZ	----------v-----A
CPZ.CD.CPZANT	----------H-------A
CPZ.GA.CPZGAB	---------L---V-----A
CPZ.US.CPZUS	--M----------V-----A

Study Subject ID:01RCH16
Study Subject Clone:
Study Subject HLA:A2,A74,B64,B45,Cw8,Cw16
Sequence: Known reactive 20Mer0: GSEELRSLYNTVATLYCVHQ p17(71–90)

Possible HLA

A2	A2.1,A*0201,A*0202,A*0203,A*0204,A*0205,A*0206,A*0207,A*0208,A*0209,A*0210,A*0211,A*0212,A*0213,A*0214,A*0216,A*0217,A*0218,A*0220,A*02
A74	A*7401,A*7402
B45	B*4501,B*5002
B64	B*1401
Cw8	Cw*08,Cw*0801,Cw*0802,C*0802,Cw*0803

Possible Epitopes based on anchor residues

(4-12)	ELRSLYNTV	A*0201
(7-15)	SLYNTVATL	A*0201
(4-12)	ELRSLYNTV	A*0202
(7-15)	SLYNTVATL	A*0202
(7-15)	SLYNTVATL	A*0204
(7-15)	SLYNTVATL	A*0205
(11-18)	TVATLYCV	A*0206
(4-12)	ELRSLYNTV	A*0214
(7-15)	SLYNTVATL	A*0214
(11-18)	TVATLYCV	A*0214

Anchor Residues Searched

A*0201	X[LM]XXXXXX[VL]
A*0201	X[LM]XXXXX[VL]
A*0201	X[LM]XXXXXXX[VL]
A*0202	X[L]XXXXXX[LV]
A*0202	X[L]XXXXX[LV]
A*0202	X[L]XXXXXXX[LV]
A*0204	X[L]XXXXXX[L]
A*0204	X[L]XXXXX[L]
A*0204	X[L]XXXXXXX[L]
A*0205	X[VLIMQ]XXXXXX[L]
A*0205	X[VLIMQ]XXXXX[L]
A*0205	X[VLIMQ]XXXXXXX[L]
A*0206	X[V]XXXXXX[V]
A*0206	X[V]XXXXX[V]
A*0206	X[V]XXXXXXX[V]
A*0207	X[L][D]XXXXX[L]

A*0207 X[L][D]XXXX[L]
A*0207 X[L][D]XXXXXX[L]
A*0214 X[VQL]XXXXXX[LV]
A*0214 X[VQL]XXXXX[LV]
A*0214 X[VQL]XXXXXXX[LV]

Study Subject ID:01RCH16
Study Subject Clone:
Study Subject HLA:A2,A74,B64,B45,Cw8,Cw16
Sequence: Known reactive 20Mer1: MFSALSEGATPQDLNTMLNT p24(39–58)

Possible HLA

A2	A2.1,A*0201,A*0202,A*0203,A*0204,A*0205,A*0206,A*0207,A*0208,A*0209,A*0210,A*0211,A*0212,A*0213,A*0214,A*0216,A*0217,A*0218,A*0220,A*02
A74	A*7401,A*7402
B45	B*4501,B*5002
B64	B*1401
Cw8	Cw*08,Cw*0801,Cw*0802,C*0802,Cw*0803

Possible Epitopes based on anchor residues

(10-17)	PQDLNTML	A*0205
(10-17)	PQDLNTML	A*0214

Anchor Residues Searched

A*0201	X[LM]XXXXXX[VL]
A*0201	X[LM]XXXXX[VL]
A*0201	X[LM]XXXXXXX[VL]
A*0202	X[L]XXXXXX[LV]
A*0202	X[L]XXXXX[LV]
A*0202	X[L]XXXXXXX[LV]
A*0204	X[L]XXXXXX[L]
A*0204	X[L]XXXXX[L]
A*0204	X[L]XXXXXXX[L]
A*0205	X[VLIMQ]XXXXXX[L]
A*0205	X[VLIMQ]XXXXX[L]
A*0205	X[VLIMQ]XXXXXXX[L]
A*0206	X[V]XXXXXX[V]
A*0206	X[V]XXXXX[V]
A*0206	X[V]XXXXXXX[V]
A*0207	X[L][D]XXXXX[L]
A*0207	X[L][D]XXXX[L]
A*0207	X[L][D]XXXXXX[L]
A*0214	X[VQL]XXXXXX[LV]
A*0214	X[VQL]XXXXX[LV]
A*0214	X[VQL]XXXXXXX[LV]

This table lists epitopes that are experimentally observed to be presented by a HLA type carried by the patient, but the de£ned epitope has substitutions relative to the peptides from your reference strains and so might be missed by your reagents: in HXB2 for Gag, Pol; MN for Env; BRU for Nef, relative to most B clade Sequences in the database:

Protein	Epitope in Database	Epitope in Ref. strain	Epitope in Consensus B	HLA	Notes
p17(77–85)	SLFNTVATL	SLYNTVATL	SLYNTVATL	A*0201	
Protease(3–11)	ITLWQRPLV	VTLWQRPLV	ITLWQRPLV	A*6802,A*7401,A19	
Protease(3–11)	ITLWQRPLV	VTLWQRPLV	ITLWQRPLV	A*7401	
RT(179–187)	VIYQYMMDL	VIYQYMDDL	VIYQYMDDL	A2	
RT(179–187)	VIYQYMMDL	VIYQYMDDL	VIYQYMDDL	A2, A*0202	
RT(308–317)	EILKEPVGHV	EILKEPVHGV	EILKEPVHGV	A*0201	
RT(436–445)	GVETFYVDGA	GAETFYVDGA	GAETFYVDGA	B45	
gp160(121–129)	KLTPLCVSL	KLTPLCVTL	KLTPLCVTL	A2	
gp160(156–165)	NCSFNISTSI	NCSFNITTSI	NCSFNITTSI	Cw*08	
gp160(156–165)	NCSFNISTSI	NCSFNITTSI	NCSFNITTSI	Cw8	
gp160(192–200)	KLTSCNTSV	RLISCNTSV	RLISCNTSV	A2	
gp160(192–200)	TLTSCNTSV	RLISCNTSV	RLISCNTSV	A2	
gp160(192–200)	TLTSCNTSV	RLISCNTSV	RLISCNTSV	A2.1	
gp160(239–247)	CTNVSTVQC	CKNVSTVQC	CTNVSTVQC	Cw8	
gp160(311–320)	RGPGRAFVTI	IGPGRAFYTT	IGPGRAFYTT	A*0201	
gp160(311–320)	RGPGRAFVTI	IGPGRAFYTT	IGPGRAFYTT	A2	
gp160(311–320)	MGPKRAFYAT	IGPGRAFYTT	IGPGRAFYTT	A2	
gp160(369–375)	PEIVTHS	PEIVMHS	PEIVMHS	A2	
gp160(377–387)	NSGGEFFYSNS	NCGGEFFYCNT	NCGGEFFYCNT	A2	
gp160(700–708)	AVLSVVNRV	AVLSIVNRV	AVLSIVNRV	A2	
gp160(747–755)	RLVNGSLAL	RLVHGFLAI	RLVDGFLAL	A2	
gp160(770–778)	RLRDLLLIV	HHRDLLLIA	RLRDLLLIV	A*0201	
gp160(813–822)	SLLNATDIAV	SLLNATAIAV	SLLNATAIAV	A*0201	
gp160(813–822)	SLLNATDIAV	SLLNATAIAV	SLLNATAIAV	A2	
gp160(813–822)	SLLNATDIAV	SLLNATAIAV	SLLNATAIAV	A2.1	
gp160(814–822)	LLNATDIAV	LLNATAIAV	LLNATAIAV	A2	
Nef(136–145)	PLTFGWCFKL	PLTFGWCYKL	PLTFGWCFKL	A2	

Table 1: **p17**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(77–85)	p17(77–85 Clade A)	SLFNTVATL	HIV-1 infection	human(A*0201)	[Dorrell (1999)]
	• Epitope SL9: CTL responses in three individuals with non-clade B infections were studied, 2 with subtype A infections, 1 with subtype C – their infections all originated in East Africa • This epitope is most commonly SLYNTVATL in B subtype, and CTL from the C subtype infection did not recognize B clade gag or the 3Y form of the epitope, but do recognize the predominant A and C clade form, SLFNTVATL				

Table 2: **Protease**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
Protease(3–11)	Protease(71–79 LAI)	ITLWQRPLV		human(A*6802,A*7401,A*0)	[Ding (1998)]
	• Predicted on binding motif, no truncations analyzed • Clade A/B/D consensus, S. Rowland-Jones, pers. comm.				
Protease(3–11)	RT(71–79 A/B/D)	ITLWQRPLV	?	human(A*7401)	[Brander & Goulder(2001)]
	• C. Brander notes this is an A*7401 epitope				

Table 3: **RT**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
RT(179–187)	RT()	VIYQYMMDL	HIV-1 exposure	human(A2)	[Rowland-Jones (1998a)]
	• A CTL response was found in exposed but uninfected prostitutes from Nairobi using previously-de£ned B clade epitopes that tended to be conserved in A and D clades – such cross-reactivity could protect against both A and D and confer protection in Nairobi where both subtypes are circulating • The A and D consensus sequences are both VIYQYMMDL				
RT(179–187)	Pol()	VIYQYMMDL	HIV-1 exposure	human(A2, A*0202)	[Rowland-Jones (1998b)]
	• HIV-speci£c CTL were found in exposed seronegative prostitutes from Nairobi – these CTL may confer protection • Seroprevalence in this cohort is 90-95% and their HIV-1 exposure is among the highest in the world • Most isolated HIV strains are clade A in Nairobi, although clades C and D are also found – B clade epitopes are often cross-reactive, however stronger responses are frequently observed using A or D clade versions of epitopes • This epitope is conserved among A, B and D clade viruses				
RT(308–317)	RT()	EILKEPVGHV	HIV-1 infection	human(A*0201)	[van der Burg (1997), Menendez-Arias (1998)]
	• Recognized by CTL from a long-term survivor, SPIETVPVKL was also recognized • Recognized by CTL from a progressor, EELRQHLLRW and TWETWWTEYW were also recognized				
RT(436–445)	Pol(591–600 IIIB)	GVETFYVDGA	HIV-1 infection	human(B45)	[Wilson (1999)]
	• This study describes maternal CTL responses in the context of mother-to-infant transmission • Detection of CTL escape mutants in the mother was associated with transmission, but the CTL-susceptible forms of the virus tended to be found in infected infants • No variants of this epitope were found in a non-transmitting mother who had a CTL response to it • This epitope spans the Pol p66 RT – p15 (RNAse) domain				

Table 4: **gp160**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
gp160(121–129)	gp120(121–129)	KLTPLCVSL	*in vitro* stimulation	human(A2)	[Zarling (1999)]
	• This study compares the ability of macrophages and dendritic cells to stimulate primary responses in CD8+ lymphocytes isolated from HLA-appropriate HIV-uninfected donors using peptide-pulsed APC – the dendritic cells performed better as APC for the stimulation of primary responses • Strong CTL responses were elicited by the epitopes DRFYKTLRA and GEIYKRWII when presented by either immature or mature dendritic cells – macrophages were not able to prime a CTL response against DRFYKTLRA • A weak response to KLTPLCVSL was stimulated using macrophages as the APC • No detectable response was observed for the following previously-de£ned HIV epitopes: KIRLRPGGK, ILKEPVHGV, IRLRPGGK, GPKVKQWPL				
gp160(156–165)	gp120(156–165)	NCSFNISTSI	HIV-1 infection	human(Cw*08)	[Ferris (1999)]
	• Recognized by CTL clone LWF A5, isolated from a lab worker exposed to HIV-1 in 1985 • The processing of this epitope is TAP1/2-dependent, as are most Env epitopes, and it contains two N-linked glycosylation sites that are glycosylated in Env • Only peptide that has been deglycosylated, a process that changes asparagine (N) to aspartic acid (D) was recognized: the aspartic acid at position 5 was critical, position 1 could be either D or N • This peptide also contains a Cys involved in a disul£de linkage but reducing conditions did not effect recognition by CTL clone LWF A5 • The HIV-1 Env epitopes are typically processed by a TAP1/2 dependent mechanism, which involves cotranslational translocation into the ER, glycosylation, export back into the cytosol, and deglycosylation for processing, and retransport into the ER for the association with class I molecules • The particular pathway of generating an epitope may have an impact on the presentation of that epitope, quantitatively as well as qualitatively				
gp160(156–165)	gp120(156–165 IIIB)	NCSFNISTSI	HIV-1 infection	human(Cw8)	[Sipsas (1997)]
	• HIV IIIB proteins were used to de£ne the range of CTL epitopes recognized by 3 lab workers accidentally infected with HIV-1 IIIB • NCSFNITTSI, a variant found in HIV-1 MN, was not recognized, thus this epitope was type-speci£c • NCSFNISTSI contains two potential N-linked glycosylation sites and cysteine residue, possibly related to the requirement for a high sensitizing dose of peptide for CTL activity				
gp160(192–200)	gp120(192–199 HXB2R)	KLTSCNTSV	HIV-1 infection	human(A2)	[Brander (1995)]
	• Epitope predicted on HLA binding motif, and studied in the context of inclusion in a synthetic vaccine				
gp160(192–200)	gp120(197–205)	TLTSCNTSV	no CTL shown	human(A2)	[Garboczi (1992)]
	• Crystallization of HLA-A2 molecules complexed with antigenic peptides – refers to Dadaglio *et al* 1991				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
gp160(192–200)	gp120(199–207)	TLTSCNTSV	peptide immunization and HIV-1 infection	human(A2.1)	[Brander (1996)]
	• This epitope was recognized by PBMC from 6/14 HIV+ asymptomatic patients • This epitope was used along with pol CTL epitope ALQDSGLEV and a tetanus toxin T helper epitope for a synthetic vaccine • This vaccine failed to induce a CTL response, although a helper response was evident				
gp160(239–247)	gp120(241–249 LAI)	CTNVSTVQC	HIV-1 infection	human(Cw8)	[Sipsas (1997)]
	• HIV IIIB proteins were used to de£ne the range of CTL epitopes recognized by 3 lab workers accidentally infected with HIV-1 IIIB • CTNVSTVQC contains a potential N-linked glycosylation site and cysteine residues, possibly related to a requirement for a high sensitizing dose of peptide for CTL activity				
gp160(311–320)	gp160(318–327 IIIB)	RGPGRAFVTI	CTL line from HIV-donor	human(A*0201)	[Alexander-Miller (1996)]
	• This immunogenic peptide does not have the known binding motif for A2.1 • The same optimal peptide for this human HLA-A2.1 epitope was observed for a murine H-2 D^d epitope				
gp160(311–320)	gp160(318–327 IIIB)	RGPGRAFVTI	vaccinia IIIB gp160	human(A2)	[Achour (1996)]
	• Individual was immunized with rec vaccinia gp160 IIIB and boosted with puri£ed gp160 • Lysis only occurs with IIIB P18 peptide pulsed onto autologous targets; MN, RF, SIMI P18 peptides fail to stimulate CTL • Restimulating immune cells from gp160 IIIB vaccinees with MN, RF, or SIMI P18 did not enhance the MN, RF, or SIMI speci£c CTL response				
gp160(311–320)	gp160(318–327 SIMI)	MGPKRAFYAT	vaccinia SIMI gp160	human(A2)	[Achour (1996)]
	• Individual was immunized with rec vaccinia gp160 SIMI and boosted with puri£ed recombinant gp160 SIMI • P18 MN and RF peptides were able to stimulate the HIV-speci£c CTL that arose in response to the SIMI vaccination, thus the P18 MN peptide (IGPGRAFYTT) and the P18 RF peptide (KGPGRVIYAT) could cross-react • The P18 IIIB peptide does not cross-react (RGPGRAFVTI in the epitope region) • gp160 SIMI primed immune cells could generate a signi£cantly broader speci£city when stimulated with P18 MN or P18RF peptides, but not P18 IIIB				
gp160(369–375)	gp120(374–380 BRU)	PEIVTHS	HIV-1 infection	human(A2)	[Dadaglio (1991)]
	• De£ned through blocking CTL activity, and Env deletions				
gp160(377–387)	gp120(377–387)	NSGGEFFYSNS		human(A2)	[Hickling (1990)]
	• Peptides recognized by class I restricted CTL can bind to class II				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
gp160(700–708)	gp41(705–714)	AVLSVVNRV	HIV-1 infection	human(A2)	[Ferris (1999)]
	• This epitope is processed by a TAP1/2 dependent mechanism				
gp160(747–755)	gp41(747–755)	RLVNGSLAL	HIV-1 infection	human(A2)	[Parker (1992)]
	• Studied in the context of HLA-A2 peptide binding				
gp160(770–778)	Env(679–777)	RLRDLLLIV	HIV-1 infection	human(A*0201)	[Kmieciak (1998)]
	• CTL responses in six patients to four Env epitopes were studied: D2: LLNATAIAV, 5.3: RLRDLLLIV, D1: KLTPLCVTL, and 4.3: QMHEDIISL – all have A2 anchor residues • The C terminal epitopes (D2 and 5.3) were highly variable and the variability was considered responsible for limited CTL response, while D1 and 4.3, N-terminal epitopes, were much more conserved and gave evidence of high levels of CTL response *in vitro* • Peptides 5.3 and D2 bound to HLA A*0201 with low af£nity and were variable, particularly D2;				
gp160(813–822)	gp41(814–823 LAI)	SLLNATDIAV	MN rec gp160	human(A*0201)	[Dupuis (1995)]
	• Of two CTL clones, one reacted only with 815-823, the other with 814-823 and 815-823 • Noted to be A*0201 in Brander *et al*., 1999 database				
gp160(813–822)	gp41(814–823)	SLLNATDIAV	HIV-1 infection	human(A2)	[Kundu (1998b)]
	• Allogeneic dendritic cells (DCs) were obtained from HLA-identical siblings, pulsed with rgp160 MN or A2-restricted HIV-1 epitope peptides, and infused monthly into six HIV-infected patients • 1/6 showed increased env-speci£c CTL and increased lymphoproliferative responses, 2/6 showed increase only in proliferative responses, and 3/6 showed no change – pulsed DCs were well tolerated • SLLNATDIAV is a conserved HLA-A2 epitope included in this study – 4/6 patients had this sequence as their HIV direct sequence, and 3 of these had a detectable CTL response – the other two had either the sequence SLFNAIDIAV or SLLNTTDIVV and no detectable CTL response • CTL demonstrated against peptide-coated target, epitope is naturally processed and enhancible with vaccine				
gp160(813–822)	Env(814–823 Clade B)	SLLNATDIAV	HIV-1 MN rgp160	human(A2.1)	[Kundu (1998a)]
	• Ten HIV-1+ HLA A2 asymptomatic individuals were given two courses of HIV-1 MN rgp160 vaccine over a 2 year period • Two hundred and £fty three HIV-1 peptides of 9 or 10 aa possessing the HLA-A2.1 binding motif (Leu at position 2, Val at the C terminus) were identi£ed in gp160, of which 25 had a high or intermediate binding af£nity • Eleven peptides were studied that had high HLA-A2 binding af£nity – a CTL response was detected to 9/11 peptides in at least 1 individual • CTL responses after reimmunization may include recall responses – only individuals with vaccine cross-reactive sequences prior to vaccination showed detectable CTL responses • CTL to overlapping peptides in this region gave a positive response in the greatest number of patients • ALTERNATIVE EPITOPES: LLNATDIAV and LLNATDIAVA – CTL were induced by vaccine in those that had the sequence SLLNATAIAVA in their own infection, but not in those with: NLLNTIAIAVA or NLFNTTAIAVA or SLLNATAITVA				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
gp160(814–822)	gp41(815–823 LAI)	LLNATDIAV	MN rec gp160	human(A2)	[Dupuis (1995)]
	• Of two CTL clones, one reacted only with 815-823, the other with 814-823 and 815-823				

Table 5: **Nef**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
Nef(136–145)	Nef(136–145)	PLTFGWCFKL	HIV-1 infection	human(A2)	[Durali (1998)]
	• Cross-clade CTL response was studied by determining the CTL activity in seven patients from Bangui, (6 A subtype, and 1 AG recombinant infections) and one A subtype infection from a person living in France originally from Togo, to different antigens expressed in vaccinia • Pol reactivity: 8/8 had CTL to A subtype, and 7/8 to B subtype, and HIV-2 Pol was not tested • Gag reactivity: 7/8 reacted with A or B subtype gag, 3/8 with HIV-2 Gag • Nef reactivity: 7/8 reacted with A subtype, and 5/8 with B subtype, none with HIV-2 Nef • Env reactivity: 3/8 reacted with A subtype, 1/8 with B subtype, none with HIV-2 Env • Patient B18 had the greatest breadth and diversity of response, and recognized Gag SLYNTVATL and Nef PLTFGWCFKL			

Table 6: **All De£ned Epitopes within the 20mer, regardless of HLA type**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(71–79)	p17(71–79 LAI)	GSEELRSLY		human(A1)	[Brander & Walker(1996)]
	• P. Goulder, pers. comm.				
p17(71–79)	p17(71–79)	GSEELRSLY	HIV-1 infection	human(A1)	[Birk (1998)]
	• A study of p17 variation considering known p17 epitopes and individuals with known HLA types revealed that p17 evolution is in¤uenced by immune pressure from CTLs				
p17(71–85)	p17(71–85 SF2)	GSEELRSLYNTVATL	HIV-1 infection	human()	[Lieberman (1997)]
	• Of 25 patients, most had CTL speci£c for more than 1 HIV-1 protein • Twelve subjects had CTL that could recognize vaccinia-expressed LAI gag • One of these 12 had CTL response to this peptide • The responding subject was HLA-A1, A11, B8, B27				
p17(74–82)	p17()	ELRSLYNTV		human(B*0801)	[Brander & Goulder(2001)]
	• Noted by Brander to be a B*0801 epitope				
p17(74–82)	p17()	ELRSLYNTV		human(B8)	[Goulder (1997c)]
	• De£ned in a study of the B8 binding motif				
p17(74–82)	p17(74–82)	ELRSLYNTV	HIV-1 infection	human(B8)	[Birk (1998)]
	• A study of p17 variation considering known p17 epitopes and individuals with known HLA types revealed that p17 evolution is in¤uenced by immune pressure from CTLs				
p17(76–86)	p17(74–86 LAI)	RSLYNTVATLY		human(A*3002)	[Brander & Goulder(2001)]
	• C. Brander notes this is an A*3002 epitope				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(76–86)	p17()	RSLYNTVATLY	HIV-1 infection	human(A*3002)	[Goulder (2000)]
	• The CTL-dominant response was focused on this epitope in a single HIV+ individual from Boston – this epitope fell outside the most recognized peptides in the study • Three peptides GSEELRSLYNTVATL (p17 residues 71-85), SALSEGATPQDLNTMLNTVG (p24 41-60), and WEKIRLRPG-GKKKYKLK(p17 16-30) contained the dominant Gag-speci£c epitope in 31 out of 44 B-clade infected individuals from Boston who showed Gag-CTL responses • Five peptides RLRPGGKKHYMIKHLVW (p17 20-36), ELRSLYNTVATLYCV (p17Gag 74-88), SALSEGATPQDLNTMLNTVG (p24 41-60), FRDYVDRFFKTLRAEQA (p24 161-177), and SILDIKQGKEPFRDY (p24 149-164) contained dominant Gag-speci£c epitopes in 32 out of 37 C-clade infected subjects from South Africa			
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A*02)	[Huang (2000)]
	• The single cell ELISPOT assay was optimized and highly speci£c, and found to work well even after the primary cells had been frozen and thawed • Increases in gamma IFN producing cells were observed in response to anti-retroviral therapy using single cell IFN-gamma-production ELISPOT • 4/8 A*02 subjects had a positive response to this epitope indicating that it is a major epitope for CD8+ gamma IFN production • In 3/3 HLA A*02, B*27 individuals, the dominant response in gag measured by both gamma IFN production and T cell lysis was a B27 epitope, p24(263-272), not the A2 SLYNTVATL epitope			
p17(77–85)	p17(77–85 HXB2)	SLYNTVATL	HIV-1 infection	human(A*0201)	[Brander (1999)]
	• Epitope SL9: Multiple natural variations in the SL9 ¤anking regions of the immunodominant epitope SLYNTVATL were tested and found not to adversely affect CTL recognition or prevent epitope processing, suggesting that viral escape from the HLA-A*0201-restricted CTL response against SLYNTVATL is probably not linked to variations in the ¤anking regions of this epitope • The substitution Y79F was an escape mutation in that it interfered with CTL recognition by one CTL clone from an A*0201 infected individual, clone 13010.B17, but it was still recognized by another CTL clone, 115.D4			
p17(77–85)	Gag()	SLYNTVATL	HIV-1 infection	human(A*0201)	[Tan (1999)]
	• Adoptive transfer of two autologous *in vitro*-expanded CTL clones against the A*0201 restricted epitopes SLYNTVATL and VIYQYMDDL were infused into a patient – they were well tolerated, but the SLYNTVATL clone was shown by tetramer staining to be rapidly eliminated through apoptosis, and the treatment had no impact upon viral load and CD4 and CD8 cell counts			

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A*0201)	[Betts (2000)]
	• Only 4/11 HLA-A2+ HIV+ individuals had CTL that reacted to SLYNTVATL, calling into question whether it is immunodominant • Ninty £ve optimally de£ned peptides from this database were used to screen for gamma interferon responses to other epitopes • Individuals that did not respond to SLYNTVATL recognized other HIV epitopes, and 2/4 SLYNTVATL responders had stronger responses to epitopes restricted by other class I alleles • SLYNTVATL was the only response detected in a one individual that was HLA A*0201, B44, B70				
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A*0201)	[Ogg (1999)]
	• Epitope SL9: CTL effector levels were measured after potent ARV therapy using HLA-tetramer complexes for the A*0201 epitopes SLYNTVATL and ILKEPVHGV in seven patients, and the B*3501 epitope DPNPQEVVL in one additional patient • Levels of CTL effectors typically decline for 5-7 days and then rebound, ¤uctuating during the £rst two weeks of therapy • After the early ¤uctuation, there was a steady exponential decay with a median half-life of 45 days				
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A*0201)	[Altman (1996)]
	• Epitope SL9: This paper introduces the tetramer methodology which permits quanti£cation of speci£c CTL based on expression of speci£c TCRs – HLA-A2 tetramers were prepared that can stain CTL lines speci£c for ILKEPVHGV and SLYNTVATL, and quantitate HIV-speci£c CD8+ cell lines in freshly isolated PBMCs • Three patients only stained the Gag epitope SLYNTVATL, one patient had the highest frequency of tetramer staining to the Pol epitope (0.77%), less to the Gag epitope (0.28%)				
p17(77–85)	Gag()	SLYNTVATL	HIV-1 infection	human(A*0201)	[Gray (1999)]
	• Administration of highly active antiretroviral therapy (HAART) reduced CD8+ cell frequency, and the CD8+ cells detected by tetramer staining were likely to be memory cells, indicating that persistently replicating viral populations are needed to maintain high frequencies of HIV-1 speci£c CTL				
p17(77–85)	p17(77–85 SF2)	SLYNTVATL	HIV-1 infection	human(A*0201)	[McAdam (1998)]
	• Epitope SL9: CTL from a patient infected with clade B virus did not recognize the clade A analog of this epitope				
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A*0201)	[Wilson (1998)]
	• Epitope SL9: HIV+ individuals were followed longitudinally using MHC tetramers in combination with 14 anti-BV chain MAbs, and clonal expansion of HIV-speci£c T cells was followed *in vivo* • Seven HIV+ people were studied, and all showed expansions of particular TCR BV clones, often several, relative to uninfected controls • Three patients were followed in detail, TCR VB expansions persisted for 2 to 3 years, with occasional transient increases • An A2-Gag speci£c line from one patient was found to be BV8, and at its highest level represented 17.5% of the patient‘s CD8+ T cells				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A*0201)	[Ogg (1998)]
	• Epitope SL9: HLA-tetrameric complexes were used in a cross-sectional study of 14 untreated HLA A*0201 positive individuals, revealing an inverse relationship between HIV Gag and Pol speci£c CTL effector cells (CTLe) and viral load • Inclusion of both the p17 SLYNTVATL and RT ILKEPVHGV epitopes gives a good representation of HLA A*0201-restricted activity • No correlation was observed between the CTLe and CD4 count or clearance rate of productively infected cells				
p17(77–85)	p17(77–85)	SLYNTVATL	none	human(A*0201)	[Walter (1997)]
	• Epitope SL9: HLA-A2 heavy chain and β2-microglobulin expressed in *E. coli* were refolded in the presence of this peptide • The HLA-A2-peptide complex elicited HLA-A2 peptide-speci£c CTL response in cells lacking HLA-A2 • Suggests that preformed HLA-peptide complexes could provide an alternate to intracellular processing for immunogens				
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A*0201)	[Lalvani (1997)]
	• Epitope SL9: A peptide-based protocol was optimized for restimulation of CTLp using optimized peptide and IL-7 concentrations – importantly this protocol does not stimulate a primary response, only secondary – peptide-speci£c CTLp counts could be obtained via staining with peptide-Class I tetramers • This peptide was one of the test peptides for optimizing the protocol				
p17(77–85)	p17(76–84)	SLYNTVATL	*in vitro* stimulation	human(A*0201)	[van der Burg (1996)]
	• Epitope SL9: Slow dissociation rate is associated with immunogenicity • CTL generated by *in vitro* stimulation of PBMC derived from uninfected individual				
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A*0201)	[Goulder (1997b), Goulder (1997a)]
	• Epitope SL9: Identical twin hemophiliac brothers were both infected with the same batch of factor VIII • One had a response to gag A2 epitope SLYNTVATL, the other to pol A2 epitope ILKEPVHGV • Viral sequencing from the twin that had no response to SLYNTVATL indicated his virus had the substituted form SLHNAVAVL • 71% of an additional set of 22 HIV-1 infected HLA-A*0201 positive donors preferentially responded to gag SLYNTVATL • Those individuals with a pol ILKEPVHGV response tended to have mutations in or around SLYNTVATL • An additional subject went from SLYNTVATL responder to non-responder coincident with a switch to the variant SLFNTVATL • [Goulder (1997a)] is a review of immune escape that summarizes this study				
p17(77–85)	Gag(77–85)	SLYNTVATL	HIV-1 infection	human(A*0201)	[Gray (1999)]
	• Epitope SL9: Peptide-tetramer complexes of A*0201 and SLYNTVATL or ILKEPVHGV were used to study individuals receiving HAART to determine the frequency of Class I HLA-restricted anti-HIV CD8+ T cells • 17/18 asymptomatic patients had a CTL response to one or both epitopes – 72% had a CTL response to SLYNTVATL • After HAART, the majority of the epitope-speci£c CTL were apparently memory cells				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(77–85)	p17(77–85 Clade A)	SLFNTVATL	HIV-1 infection	human(A*0201)	[Dorrell (1999)]
	• Epitope SL9: CTL responses in three individuals with non-clade B infections were studied, 2 with subtype A infections, 1 with subtype C – their infections all originated in East Africa • This epitope is most commonly SLYNTVATL in B subtype, and CTL from the C subtype infection did not recognize B clade gag or the 3Y form of the epitope, but do recognize the predominant A and C clade form, SLFNTVATL				
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A*0201)	[Brander (1998)]
	• Epitope SL9: Of 17 infected HLA A*0201 subjects, 13 had CTL responses against the p17 SLYNTVATL epitope, six recognized ILKEPVHGV and £ve recognized VIYQYMDDL, and there was no correlation between viral load and recognition of a speci£c epitope • Only one subject had CTL against all three epitopes • There was signi£cant heterogeneity in the CTL response to this immunodominant epitope • The overall variation in this epitope among the 17 who had a CTL response and 11 non-HLA A*0201 HIV-1+ individuals was similar, suggesting a lack of immune pressure • Subjects were part of the San Francisco City Clinic Cohort, the ARIEL project and from the Boston area				
p17(77–85)	p17(77–85 HXB2)	SLYNTVATL	HIV-1 infection	human(A*0201)	[Hay (1999)]
	• Epitope SL9: CTL response to IPRRIRQGL was the immunodominant response in a rapid progressor – there was a subdominant response to SPAIFQSSM in Pol, and interestingly, no response to commonly immunodominant HLA A*0201 epitope SLYNTVATL, although this individual was HLA A*0201 • The individual showed a strong initial CTL response at the time of the initial drop in viremia, but it was quickly lost, although memory cells persisted • Despite the initial narrow response to two epitopes, no other CTL responses developed • No HIV-speci£c lymphoproliferative responses were detected in this patient, and neutralizing antibody response was weak • A variant of this epitope was observed *in vivo* (–F—-V-), but this mutation is recognized by SLYNTVATL-speci£c CTL, and in this case the patient's cells could present the peptide to SLYNTVATL-speci£c CTL				
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A*0201)	[Kalams (1999)]
	• Two patients were followed before and after HAART – reduced plasma HIV-1 RNA levels resulted in a decline in HIV-speci£c in-vivo activated CTL such that by day 260 CTL activities were undetectable • ERYLKDQQL was the dominant response in one of the individuals, SLYNTVATL subdominant • Sporadic breakthrough in viremia resulted in transient increases in CTLp • Memory CTL frequency directed against Vac-Gag, Vac-RT, Vac-Env, and Vac-Nef initially increased with HAART and then decreased with the decline of the viral load				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(77–85)	Gag(77–85)	SLYNTVATL	HIV-1 infection	human(A*0201)	[Spiegel (2000)]
	• High levels of CD8+ HIV-1 speci£c and cytomegalovirus speci£c CTL were detected by HLA-A*0201-peptide tetramers in 3 infected subjects with very low CD4 counts, but CD8 T cell mediated effector activity was not seen • Thus HIV-1 speci£c CD8+ cells may be present but may lack direct effector activity in late disease, suggesting that overcoming antigen unresponsiveness may be a useful therapeutic strategy				
p17(77–85)	Gag(77–85)	SLYNTVATL	HIV-1 infection	human(A*0201)	[Larsson (1999)]
	• ELISPOT was used to assay the CD8 T cell response to the HIV-1 proteins Gag, Pol, Nef or Env expressed in vaccinia vectors in 19 HIV+ people • The highest CTL frequency was directed at epitopes Pol • In A*0201 individuals, higher numbers of spot-forming T cells were directed against HIV-1 proteins expressed in vaccinia than to peptides SLYNTVATL and ILKEPVHGV presented by A2				
p17(77–85)	p17()	SLYNTVATL	HIV-1 infection	human(A*0201)	[Goulder (2000)]
	• The CTL-dominant response was focused on this epitope in 11/25 HLA A2 (A*0201 or A*0202) HIV+ individuals from Boston and in 1/8 HLA A2 HIV+ individuals from Durban • Three peptides GSEELRSLYNTVATL (p17 residues 71-85), SALSEGATPQDLNTMLNTVG (p24 41-60), and WEKIRLRPG-GKKKYKLK(p17 16-30) contained the dominant Gag-speci£c epitope in 31 out of 44 B-clade infected individuals from Boston who showed Gag-CTL responses • Five peptides RLRPGGKKHYMIKHLVW (p17 20-36), ELRSLYNTVATLYCV (p17Gag 74-88), SALSEGATPQDLNTMLNTVG (p24 41-60), FRDYVDRFFKTLRAEQA (p24 161-177), and SILDIKQGKEPFRDY (p24 149-164) contained dominant Gag-speci£c epitopes in 32 out of 37 C-clade infected subjects from South Africa			
p17(77–85)	p17(77–85 LAI)	SLYNTVATL		human(A*0201)	[Brander & Goulder(2001)]
	• C. Brander notes this is an A*0201 epitope				
p17(77–85)	p17(77–85)	SLYNTVATL		human(A*0202)	[Brander & Goulder(2001)]
	• C. Brander notes that this epitope can be presented by A*0201 and A*0202				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(77–85)	p17()	SLYNTVATL	HIV-1 infection	human(A*0202)	[Goulder (2000)]
	• The CTL-dominant response was focused on this epitope in 11/25 HLA A2 (A*0201 or A*0202) HIV+ individuals from Boston and in 1/8 HLA A2 HIV+ individuals from Durban • Three peptides GSEELRSLYNTVATL (p17 residues 71-85), SALSEGATPQDLNTMLNTVG (p24 41-60), and WEKIRLRPG-GKKKYKLK(p17 16-30) contained the dominant Gag-speci£c epitope in 31 out of 44 B-clade infected individuals from Boston who showed Gag-CTL responses • Five peptides RLRPGGKKHYMIKHLVW (p17 20-36), ELRSLYNTVATLYCV (p17Gag 74-88), SALSEGATPQDLNTMLNTVG (p24 41-60), FRDYVDRFFKTLRAEQA (p24 161-177), and SILDIKQGKEPFRDY (p24 149-164) contained dominant Gag-speci£c epitopes in 32 out of 37 C-clade infected subjects from South Africa			
p17(77–85)	p17(77–85 LAI)	SLYNTVATL		human(A*0205)	[Brander & Goulder(2001)]
	• C. Brander notes that this epitope can be presented by A*0201 and A*0202				
p17(77–85)	p17()	SLYNTVATL	HIV-1 exposed seronegative	human(A*0214,A*0201)	[Kaul (2000)]
	• 11/16 heavily HIV exposed but persistently seronegative sex-workers in Nairobi had HIV-speci£c CD8 gamma-IFN responses in the cervix – systemic CD8+ T cell responses tended to be to the same epitopes but at generally lower levels than cervical CD8+ T cell responses • Low risk individuals did not have such CD8+ cells • CD8+ epitopes T cell DTVLEDINL (3 individuals), SLYNVATL (4 individuals), LSPRTLNAW (3 individuals) and YPLTFGWCF (4 individuals) were most commonly recognized by the HIV-resistant women • The epitope variants SLYNTVATL and SLFNTVATL were both recognized			
p17(77–85)	Gag(77–85)	SLYNTVATL	HIV A2-polyepitope (polytope) DNA vaccine with vaccinia boost (rVV.HIV.pt)	human(A2)	[Woodberry (1999)]
	• A polyepitope vaccine was generated in a vaccinia construct that contiguously encoded seven epitopes, all presented by HLA A-2 • HHD mice have a transgene of HLA A2 linked to the transmembrane and cytotoxic domains of H-2D^d – this transgene is the only MHC molecule expressed in the mice • CTL responses to Gag (77-85) SLYNTVATL, Pol (476-484) ILKEPVHGV, gp120 (120-128) KLTPLCVTL, and Nef (190-198) AFHHVAREL were observed in HIV polytope HHD-vaccinated mice, and these responses were enhanced with vaccinia boost • No CTL immune responses were generated against HLA A2-restricted HIV epitopes Nef 157-166 (PLTFGWCYKL), Pol 346-354 (VIYQYMDDL), and Nef 180-189 (VLEWRFDSRL) • Sixteen HLA A2+ patients were tested for their ability to make CTL responses by peptide restimulation in culture with the epitopes selected for inclusion in the polytope – one individual recognized all seven of these epitopes; 7 patients had CTL cultures able to recognize at least one of the epitopes, and 6 of those 7 recognized more than one epitope, but they were not able to test all peptides for all patients; many patients only had three peptides tested • SLYNTVATL was recognized by 5/16 HLA-A2 patients			

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(77–85)	p17(77–85)	SLYNTVATL	Live recombinant canarypox (CP) virus vaccine containing multiple HIV-1 genes (HIV-1 MN gp120, HIV-1 LAI gp41, HIV-1 LAI Gag, HIV-1 LAI protease)	human(A2)	[Carruth (1999)]
	• CD4+ and CD8+ Gag and Env speci£c CTL responses were detected in only 1/5 vaccinated volunteers, and were not detectable 1 year after vaccination • CTL responses to epitopes SLYNTVATL and TVYYGVPVWK from HIV+ control patients were used as positive controls • The study explored why vaccinees were non-responsive – non-response was not due to inherent defects or differences in the ability of these individuals to process and present antigen • Lack of response to SLYNTVATL led the authors to speculate that the immunodominance of this epitope in natural infections may not be recapitulated by vaccine antigen				
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A2)	[Birk (1998)]
	• Epitope SL9: A study of p17 variation considering known p17 epitopes and individuals with known HLA types revealed that p17 evolution is in¤uenced by immune pressure from CTLs				
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A2)	[Callan (1998)]
	• Epitope SL9: Included as a negative control in a tetramer study of A2-EBV CTL response				
p17(77–85)	p17()	SLYNTVATL	HIV-1 infection	human(A2)	[Wagner (1998)]
	• Epitope SL9: CTL speci£c for HIV epitopes were used to show that the mediators of both the cytolytic (granzyme A was used as the marker) and non-cytolytic (HIV-1 inhibitory chemokines MIP-1 α and RANTES were used as markers) anti-viral responses are localized within the CTL‘s cytotoxic granules				
p17(77–85)	p17(77–85 HXB2)	SLYNTVATL	HIV-1 infection	human(A2)	[Collins (1998)]
	• Epitope SL9: Two CTL clones recognize this epitope, but not the NL4-3 form of the epitope SLYNTIAVL • Nef down-regulates MHC class I molecules, which inhibits CTL killing, and this down-regulation can be partially compensated for by adding excess soluble peptide				
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A2)	[Durali (1998)]
	• Epitope SL9: Cross-clade CTL response was studied by determining the CTL activity in seven patients from Bangui, (6 A subtype, and 1 AG recombinant infections) and one A subtype infection from a person living in France originally from Togo, to different antigens expressed in vaccinia • Pol reactivity: 8/8 had CTL to A subtype, and 7/8 to B subtype, and HIV-2 Pol was not tested • Gag reactivity: 7/8 reacted with A or B subtype gag, 3/8 with HIV-2 Gag • Nef reactivity: 7/8 reacted with A subtype, and 5/8 with B subtype, none with HIV-2 Nef • Env reactivity: 3/8 reacted with A subtype, 1/8 with B subtype, none with HIV-2 Env • Patient B18 had the greatest breadth and diversity of response, and recognized Gag SLYNTVATL and Nef PLTFGWCFKL				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A2)	[Kundu (1998b)]
	• Epitope SL9: Allogeneic dendritic cells (DCs) were obtained from HLA-identical siblings, pulsed with rgp160 MN or A2-restricted HIV-1 epitope peptides, and infused monthly into six HIV-infected patients • 1/6 showed increased env-speci£c CTL and increased lymphoproliferative responses, 2/6 showed increase only in proliferative responses, and 3/6 showed no change – pulsed DCs were well tolerated • SLYNTVATL is a conserved HLA-A2 epitope included in this study – 3/6 patients had this sequence as their HIV direct sequence, one had the form SLYNTVAVL and all four of these had a detectable CTL response – the other two had either the sequence SLFSAVAVL or SLFSAVAAL and no detectable CTL response			
p17(77–85)	p17(77–85 IIIB)	SLYNTVATL	HIV-1 infection	human(A2)	[Sipsas (1997)]
	• Epitope SL9: HIV IIIB proteins were used to de£ne the range of CTL epitopes recognized by 3 lab workers accidentally infected with HIV-1 IIIB • SLYNTVAVL, a variant found in HIV-1 MANC, was also recognized • SLFNTVAVL, a variant found in HIV-1 NY5CG, was also recognized				
p17(77–85)	p17()	SLYNTVATL	HIV-1 infection	human(A2)	[Rowland-Jones (1998a)]
	• Epitope SL9: A CTL response was found in exposed but uninfected prostitutes from Nairobi using previously-de£ned B clade epitopes that tended to be conserved in A and D clades – such cross-reactivity could protect against both A and D and confer protection in Nairobi where both subtypes are circulating • The A subtype consensus is SLfNtvatL • The D subtype consensus is SLyNTvATL				
p17(77–85)	p17()	SLYNTVATL	HIV-1 infection	human(A2)	[Sewell (1997)]
	• Epitope SL9: Naturally occurring variants of this epitope escaped killing and acted as antagonists • The following variants were found in HIV-1 infected patients who mounted a strong response against this epitope: –F——, –F—-V-, –S——, -SF——, –L——, ——I—, ——I-V-, –F–I—, –F–I-V-, –F-A—- • All variants bound to A2 with at least half the af£nity of SLYNTVATL except the triple mutant: –F–I-V- • Antagonism could be observed at low concentrations, abrogating lysis at an antagonist:agonist ratio of 1:10 – the antagonism was observed in one SLYNTVATL-speci£c CTL line but not another				
p17(77–85)	p17(77–85 HXB2)	SLYNTVATL	HIV-1 infection	human(A2)	[Yang (1997b)]
	• Epitope SL9: A chimeric universal T cell receptor was created by linking CD4 or an HIV-speci£c anti-gp41 Ig sequence to the signaling domain of the T cell receptor chain ζ, and transduced into CD8+ cells • The response using universal-receptor-bearing CD8+ cells to lyse infected cells *in vitro* was comparable to the natural occurring responses of CTL-clones from HIV+ individuals in terms of kinetics and ef£ciency • A CTL clone speci£c for this epitope was used for the comparison				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(77–85)	p17(77–85)	SLYNTVATL	*in vitro* stimulation	human(A2)	[Stuhler & Schlossman(1997)]
	• Epitope SL9: Keyhole limpit hemocyanin or tetanus toxoid Th epitope co-expression with peptide CTL epitopes on the same APC was required for induction of peptide-speci£c CTL				
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A2)	[Yang (1996)]
	• Epitope SL9: CD4+ cell lines acutely infected with HIV were studied to determine their susceptibility to lysis by CTL • Clones speci£c for RT lysed HIV-1 infected cells at lower levels than Env or Gag speci£c clones • The distinction was thought to be due to lower expression of RT relative to Env and Gag • CTL can lyse infected cells early after infection, possibly prior to viral production				
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A2)	[Yang (1997a)]
	• Epitope SL9: CTL inhibit HIV-1 replication at effector cell concentrations comparable to those found *in vivo* • CTL produced HIV-1-suppressive soluble factors – MIP-1α, MIP-1β, RANTES, after antigen-speci£c activation • CTL suppress HIV replication more ef£ciently in HLA-matched cells				
p17(77–85)	p17(77–85 LAI)	SLYNTVATL	HIV-1 infection	human(A2)	[Parker (1992), Parker (1994)]
	• Epitope SL9: Examined in the context of motifs important for HLA-A2 binding				
p17(77–85)	p17(77–85 LAI)	SLYNTVATL	HIV-1 infection	human(A2)	[McMichael & Walker(1994)]
	• Epitope SL9: Review of HIV CTL epitopes				
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A2)	[Tsomides (1994)]
	• Epitope SL9: CTL clones recognize naturally processed peptide				
p17(77–85)	p17(77–85)	SLYNTVATL	Peptide stimulation *in vitro*	human(A2)	[Stuhler & Schlossman(1997)]
	• Epitope SL9: A three cell-type cluster consisting of APCs, Th, and CTLs is the minimal regulatory unit required for Th cell-dependent induction of CTLs				
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A2)	[Cao (1997)]
	• Epitope SL9: The consensus peptides of B and D clade viruses and some Cs have the sequence SLYNTVATL • The consensus peptide of A, and some C strains have SLFNTVATL, a form that is cross-reactive				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(77–85)	Gag(77–85)	SLYNTVATL	HIV-1 infection	human(A2)	[Dyer (1999)]
• Epitope SL9: CTL speci£c responses were measured over a 1.3 to 1.5 year period in members of the Sydney Blood Bank Cohort (SBBC) who had been infected with a natural attenuated strain of HIV-1 which was Nef-defective • Some of these patients had prolonged high levels of CTL effector and memory cells despite low viral load					
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A2)	[Harrer (1998)]
• Epitope SL9: Two overlapping epitopes were recognized in a long-term survivor, restricted by two different HLA molecules, HLA-A11(TLYCVHQR) and -A2 (SLYNTVATL) • Viral sequence substitutions were present in this individual which did not affect viral replication and did not alter CTL-recognition of the A2 epitope, but reduced recognition of the A11 epitope, indicative of immune escape					
p17(77–85)	p17()	SLYNTVATL	HIV-1 exposure	human(A2, A*0202)	[Rowland-Jones (1998b)]
• Epitope SL9: HIV-speci£c CTL were found in exposed seronegative prostitutes from Nairobi – these CTL may confer protection • Seroprevalence in this cohort is 90-95% and their HIV-1 exposure is among the highest in the world • Most isolated HIV strains are clade A in Nairobi, although clades C and D are also found – B clade epitopes are often cross-reactive, however stronger responses are frequently observed using A or D clade versions of epitopes • This epitope is conserved among B and D clade viruses • The Clade A version of the epitope, SLFNTVATL, was preferentially recognized by CTL • This epitope was recognized by two different exposed seronegative prostitutes					
p17(77–85)	p17()	SLYNTVATL	HIV-1 infection	human(B*0201)	[Wilson (2000)]
• Three individuals with highly focused HIV-speci£c CTL responses were studied during acute infection using tetramers – high frequencies of HIV-1-speci£c CD8+ T cells were found prior to seroconversion, and there was a close temporal relationship between the number of circulating HIV-speci£c T cells and viral load was also found • All three patients were B*2705, with HLA alleles: A1, A30/31, B*2705, B35; A1, A*0301, B7, B2705; and A*0201, A*0301, B2705, B39 • ELISPOT was used to test a panel of CTL epitopes that had been de£ned earlier and were appropriate for the HLA haplotypes of the study subjects – 3/3 subjects showed a dominant response to the B*2705 epitope KRWIILGGLNK • The subject with A*0201 had a moderatly strong response to SLYNTVATL • Weak responses were observed to A*301-RLRPGGKKK, A*301-QVPLRPMTYK, and B7-TPGPGVRYPL in the subject who was HLA A1, A*0301, B7, B*2705 • No acute response was detected to the following epitopes: A*201-ILKEPVHGV, A*301-KIRLRPGGK, A*301-AIFQSSMTK, A*301-TVYYGVPVWK, B35-EPIVGAETF, B35-HPDIVIYQY, B35-PPIPVGEIY, B35-NSSKVSQNY, B35-VPLRPMTY, B35-DPNPQEVVL			

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(B62)	[Goulder (1997a)]

- Epitope SL9: This paper is a review of CTL and immune evasion, but it presents a study of a shift from an HLA-A*0201 response to SLYNTVATL, to a B62 response to GLNKIVRMY
- As long as a strong CTL response to SLYNTVATL was evident, the epitope variants SLFNTVATL or SLYNTIATL dominated the viral population – eventually the CTL response to the index peptide became undetectable, the CTL response shifted to a focus on GLNKIVRMY, and the index peptide SLYNTVATL once again established itself as the dominant form

Table 7: **All De£ned Epitopes within the 20mer, regardless of HLA type**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(71–79)	p17(71–79 LAI)	GSEELRSLY		human(A1)	[Brander & Walker(1996)]
	• P. Goulder, pers. comm.				
p17(71–79)	p17(71–79)	GSEELRSLY	HIV-1 infection	human(A1)	[Birk (1998)]
	• A study of p17 variation considering known p17 epitopes and individuals with known HLA types revealed that p17 evolution is in¤uenced by immune pressure from CTLs				
p17(71–85)	p17(71–85 SF2)	GSEELRSLYNTVATL	HIV-1 infection	human()	[Lieberman (1997)]
	• Of 25 patients, most had CTL speci£c for more than 1 HIV-1 protein • Twelve subjects had CTL that could recognize vaccinia-expressed LAI gag • One of these 12 had CTL response to this peptide • The responding subject was HLA-A1, A11, B8, B27				
p17(74–82)	p17()	ELRSLYNTV		human(B*0801)	[Brander & Goulder(2001)]
	• Noted by Brander to be a B*0801 epitope				
p17(74–82)	p17()	ELRSLYNTV		human(B8)	[Goulder (1997c)]
	• De£ned in a study of the B8 binding motif				
p17(74–82)	p17(74–82)	ELRSLYNTV	HIV-1 infection	human(B8)	[Birk (1998)]
	• A study of p17 variation considering known p17 epitopes and individuals with known HLA types revealed that p17 evolution is in¤uenced by immune pressure from CTLs				
p17(76–86)	p17(74–86 LAI)	RSLYNTVATLY		human(A*3002)	[Brander & Goulder(2001)]
	• C. Brander notes this is an A*3002 epitope				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(76–86)	p17()	RSLYNTVATLY	HIV-1 infection	human(A*3002)	[Goulder (2000)]
	• The CTL-dominant response was focused on this epitope in a single HIV+ individual from Boston – this epitope fell outside the most recognized peptides in the study • Three peptides GSEELRSLYNTVATL (p17 residues 71-85), SALSEGATPQDLNTMLNTVG (p24 41-60), and WEKIRLRPG-GKKKYKLK(p17 16-30) contained the dominant Gag-speci£c epitope in 31 out of 44 B-clade infected individuals from Boston who showed Gag-CTL responses • Five peptides RLRPGGKKHYMIKHLVW (p17 20-36), ELRSLYNTVATLYCV (p17Gag 74-88), SALSEGATPQDLNTMLNTVG (p24 41-60), FRDYVDRFFKTLRAEQA (p24 161-177), and SILDIKQGKEPFRDY (p24 149-164) contained dominant Gag-speci£c epitopes in 32 out of 37 C-clade infected subjects from South Africa			
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A*02)	[Huang (2000)]
	• The single cell ELISPOT assay was optimized and highly speci£c, and found to work well even after the primary cells had been frozen and thawed • Increases in gamma IFN producing cells were observed in response to anti-retroviral therapy using single cell IFN-gamma-production ELISPOT • 4/8 A*02 subjects had a positive response to this epitope indicating that it is a major epitope for CD8+ gamma IFN production • In 3/3 HLA A*02, B*27 individuals, the dominant response in gag measured by both gamma IFN production and T cell lysis was a B27 epitope, p24(263-272), not the A2 SLYNTVATL epitope			
p17(77–85)	p17(77–85 HXB2)	SLYNTVATL	HIV-1 infection	human(A*0201)	[Brander (1999)]
	• Epitope SL9: Multiple natural variations in the SL9 ¤anking regions of the immunodominant epitope SLYNTVATL were tested and found not to adversely affect CTL recognition or prevent epitope processing, suggesting that viral escape from the HLA-A*0201-restricted CTL response against SLYNTVATL is probably not linked to variations in the ¤anking regions of this epitope • The substitution Y79F was an escape mutation in that it interfered with CTL recognition by one CTL clone from an A*0201 infected individual, clone 13010.B17, but it was still recognized by another CTL clone, 115.D4			
p17(77–85)	Gag()	SLYNTVATL	HIV-1 infection	human(A*0201)	[Tan (1999)]
	• Adoptive transfer of two autologous *in vitro*-expanded CTL clones against the A*0201 restricted epitopes SLYNTVATL and VIYQYMDDL were infused into a patient – they were well tolerated, but the SLYNTVATL clone was shown by tetramer staining to be rapidly eliminated through apoptosis, and the treatment had no impact upon viral load and CD4 and CD8 cell counts			

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A*0201)	[Betts (2000)]
	• Only 4/11 HLA-A2+ HIV+ individuals had CTL that reacted to SLYNTVATL, calling into question whether it is immunodominant • Ninty £ve optimally de£ned peptides from this database were used to screen for gamma interferon responses to other epitopes • Individuals that did not respond to SLYNTVATL recognized other HIV epitopes, and 2/4 SLYNTVATL responders had stronger responses to epitopes restricted by other class I alleles • SLYNTVATL was the only response detected in a one individual that was HLA A*0201, B44, B70				
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A*0201)	[Ogg (1999)]
	• Epitope SL9: CTL effector levels were measured after potent ARV therapy using HLA-tetramer complexes for the A*0201 epitopes SLYNTVATL and ILKEPVHGV in seven patients, and the B*3501 epitope DPNPQEVVL in one additional patient • Levels of CTL effectors typically decline for 5-7 days and then rebound, ¤uctuating during the £rst two weeks of therapy • After the early ¤uctuation, there was a steady exponential decay with a median half-life of 45 days				
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A*0201)	[Altman (1996)]
	• Epitope SL9: This paper introduces the tetramer methodology which permits quanti£cation of speci£c CTL based on expression of speci£c TCRs – HLA-A2 tetramers were prepared that can stain CTL lines speci£c for ILKEPVHGV and SLYNTVATL, and quantitate HIV-speci£c CD8+ cell lines in freshly isolated PBMCs • Three patients only stained the Gag epitope SLYNTVATL, one patient had the highest frequency of tetramer staining to the Pol epitope (0.77%), less to the Gag epitope (0.28%)				
p17(77–85)	Gag()	SLYNTVATL	HIV-1 infection	human(A*0201)	[Gray (1999)]
	• Administration of highly active antiretroviral therapy (HAART) reduced CD8+ cell frequency, and the CD8+ cells detected by tetramer staining were likely to be memory cells, indicating that persistently replicating viral populations are needed to maintain high frequencies of HIV-1 speci£c CTL				
p17(77–85)	p17(77–85 SF2)	SLYNTVATL	HIV-1 infection	human(A*0201)	[McAdam (1998)]
	• Epitope SL9: CTL from a patient infected with clade B virus did not recognize the clade A analog of this epitope				
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A*0201)	[Wilson (1998)]
	• Epitope SL9: HIV+ individuals were followed longitudinally using MHC tetramers in combination with 14 anti-BV chain MAbs, and clonal expansion of HIV-speci£c T cells was followed *in vivo* • Seven HIV+ people were studied, and all showed expansions of particular TCR BV clones, often several, relative to uninfected controls • Three patients were followed in detail, TCR VB expansions persisted for 2 to 3 years, with occasional transient increases • An A2-Gag speci£c line from one patient was found to be BV8, and at its highest level represented 17.5% of the patient‘s CD8+ T cells				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A*0201)	[Ogg (1998)]
	• Epitope SL9: HLA-tetrameric complexes were used in a cross-sectional study of 14 untreated HLA A*0201 positive individuals, revealing an inverse relationship between HIV Gag and Pol speci£c CTL effector cells (CTLe) and viral load • Inclusion of both the p17 SLYNTVATL and RT ILKEPVHGV epitopes gives a good representation of HLA A*0201-restricted activity • No correlation was observed between the CTLe and CD4 count or clearance rate of productively infected cells				
p17(77–85)	p17(77–85)	SLYNTVATL	none	human(A*0201)	[Walter (1997)]
	• Epitope SL9: HLA-A2 heavy chain and β2-microglobulin expressed in *E. coli* were refolded in the presence of this peptide • The HLA-A2-peptide complex elicited HLA-A2 peptide-speci£c CTL response in cells lacking HLA-A2 • Suggests that preformed HLA-peptide complexes could provide an alternate to intracellular processing for immunogens				
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A*0201)	[Lalvani (1997)]
	• Epitope SL9: A peptide-based protocol was optimized for restimulation of CTLp using optimized peptide and IL-7 concentrations – importantly this protocol does not stimulate a primary response, only secondary – peptide-speci£c CTLp counts could be obtained via staining with peptide-Class I tetramers • This peptide was one of the test peptides for optimizing the protocol				
p17(77–85)	p17(76–84)	SLYNTVATL	*in vitro* stimulation	human(A*0201)	[van der Burg (1996)]
	• Epitope SL9: Slow dissociation rate is associated with immunogenicity • CTL generated by *in vitro* stimulation of PBMC derived from uninfected individual				
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A*0201)	[Goulder (1997b), Goulder (1997a)]
	• Epitope SL9: Identical twin hemophiliac brothers were both infected with the same batch of factor VIII • One had a response to gag A2 epitope SLYNTVATL, the other to pol A2 epitope ILKEPVHGV • Viral sequencing from the twin that had no response to SLYNTVATL indicated his virus had the substituted form SLHNAVAVL • 71% of an additional set of 22 HIV-1 infected HLA-A*0201 positive donors preferentially responded to gag SLYNTVATL • Those individuals with a pol ILKEPVHGV response tended to have mutations in or around SLYNTVATL • An additional subject went from SLYNTVATL responder to non-responder coincident with a switch to the variant SLFNTVATL • [Goulder (1997a)] is a review of immune escape that summarizes this study				
p17(77–85)	Gag(77–85)	SLYNTVATL	HIV-1 infection	human(A*0201)	[Gray (1999)]
	• Epitope SL9: Peptide-tetramer complexes of A*0201 and SLYNTVATL or ILKEPVHGV were used to study individuals receiving HAART to determine the frequency of Class I HLA-restricted anti-HIV CD8+ T cells • 17/18 asymptomatic patients had a CTL response to one or both epitopes – 72% had a CTL response to SLYNTVATL • After HAART, the majority of the epitope-speci£c CTL were apparently memory cells				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(77–85)	p17(77–85 Clade A)	SLFNTVATL	HIV-1 infection	human(A*0201)	[Dorrell (1999)]
	• Epitope SL9: CTL responses in three individuals with non-clade B infections were studied, 2 with subtype A infections, 1 with subtype C – their infections all originated in East Africa • This epitope is most commonly SLYNTVATL in B subtype, and CTL from the C subtype infection did not recognize B clade gag or the 3Y form of the epitope, but do recognize the predominant A and C clade form, SLFNTVATL				
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A*0201)	[Brander (1998)]
	• Epitope SL9: Of 17 infected HLA A*0201 subjects, 13 had CTL responses against the p17 SLYNTVATL epitope, six recognized ILKEPVHGV and £ve recognized VIYQYMDDL, and there was no correlation between viral load and recognition of a speci£c epitope • Only one subject had CTL against all three epitopes • There was signi£cant heterogeneity in the CTL response to this immunodominant epitope • The overall variation in this epitope among the 17 who had a CTL response and 11 non-HLA A*0201 HIV-1+ individuals was similar, suggesting a lack of immune pressure • Subjects were part of the San Francisco City Clinic Cohort, the ARIEL project and from the Boston area				
p17(77–85)	p17(77–85 HXB2)	SLYNTVATL	HIV-1 infection	human(A*0201)	[Hay (1999)]
	• Epitope SL9: CTL response to IPRRIRQGL was the immunodominant response in a rapid progressor – there was a subdominant response to SPAIFQSSM in Pol, and interestingly, no response to commonly immunodominant HLA A*0201 epitope SLYNTVATL, although this individual was HLA A*0201 • The individual showed a strong initial CTL response at the time of the initial drop in viremia, but it was quickly lost, although memory cells persisted • Despite the initial narrow response to two epitopes, no other CTL responses developed • No HIV-speci£c lymphoproliferative responses were detected in this patient, and neutralizing antibody response was weak • A variant of this epitope was observed *in vivo* (–F—-V-), but this mutation is recognized by SLYNTVATL-speci£c CTL, and in this case the patient's cells could present the peptide to SLYNTVATL-speci£c CTL				
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A*0201)	[Kalams (1999)]
	• Two patients were followed before and after HAART – reduced plasma HIV-1 RNA levels resulted in a decline in HIV-speci£c in-vivo activated CTL such that by day 260 CTL activities were undetectable • ERYLKDQQL was the dominant response in one of the individuals, SLYNTVATL subdominant • Sporadic breakthrough in viremia resulted in transient increases in CTLp • Memory CTL frequency directed against Vac-Gag, Vac-RT, Vac-Env, and Vac-Nef initially increased with HAART and then decreased with the decline of the viral load				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(77–85)	Gag(77–85)	SLYNTVATL	HIV-1 infection	human(A*0201)	[Spiegel (2000)]
	• High levels of CD8+ HIV-1 speci£c and cytomegalovirus speci£c CTL were detected by HLA-A*0201-peptide tetramers in 3 infected subjects with very low CD4 counts, but CD8 T cell mediated effector activity was not seen • Thus HIV-1 speci£c CD8+ cells may be present but may lack direct effector activity in late disease, suggesting that overcoming antigen unresponsiveness may be a useful therapeutic strategy				
p17(77–85)	Gag(77–85)	SLYNTVATL	HIV-1 infection	human(A*0201)	[Larsson (1999)]
	• ELISPOT was used to assay the CD8 T cell response to the HIV-1 proteins Gag, Pol, Nef or Env expressed in vaccinia vectors in 19 HIV+ people • The highest CTL frequency was directed at epitopes Pol • In A*0201 individuals, higher numbers of spot-forming T cells were directed against HIV-1 proteins expressed in vaccinia than to peptides SLYNTVATL and ILKEPVHGV presented by A2				
p17(77–85)	p17()	SLYNTVATL	HIV-1 infection	human(A*0201)	[Goulder (2000)]
	• The CTL-dominant response was focused on this epitope in 11/25 HLA A2 (A*0201 or A*0202) HIV+ individuals from Boston and in 1/8 HLA A2 HIV+ individuals from Durban • Three peptides GSEELRSLYNTVATL (p17 residues 71-85), SALSEGATPQDLNTMLNTVG (p24 41-60), and WEKIRLRPG-GKKKYKLK(p17 16-30) contained the dominant Gag-speci£c epitope in 31 out of 44 B-clade infected individuals from Boston who showed Gag-CTL responses • Five peptides RLRPGGKKHYMIKHLVW (p17 20-36), ELRSLYNTVATLYCV (p17Gag 74-88), SALSEGATPQDLNTMLNTVG (p24 41-60), FRDYVDRFFKTLRAEQA (p24 161-177), and SILDIKQGKEPFRDY (p24 149-164) contained dominant Gag-speci£c epitopes in 32 out of 37 C-clade infected subjects from South Africa			
p17(77–85)	p17(77–85 LAI)	SLYNTVATL		human(A*0201)	[Brander & Goulder(2001)]
	• C. Brander notes this is an A*0201 epitope				
p17(77–85)	p17(77–85)	SLYNTVATL		human(A*0202)	[Brander & Goulder(2001)]
	• C. Brander notes that this epitope can be presented by A*0201 and A*0202				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(77–85)	p17()	SLYNTVATL	HIV-1 infection	human(A*0202)	[Goulder (2000)]
	• The CTL-dominant response was focused on this epitope in 11/25 HLA A2 (A*0201 or A*0202) HIV+ individuals from Boston and in 1/8 HLA A2 HIV+ individuals from Durban • Three peptides GSEELRSLYNTVATL (p17 residues 71-85), SALSEGATPQDLNTMLNTVG (p24 41-60), and WEKIRLRPG-GKKKYKLK(p17 16-30) contained the dominant Gag-speci£c epitope in 31 out of 44 B-clade infected individuals from Boston who showed Gag-CTL responses • Five peptides RLRPGGKKHYMIKHLVW (p17 20-36), ELRSLYNTVATLYCV (p17Gag 74-88), SALSEGATPQDLNTMLNTVG (p24 41-60), FRDYVDRFFKTLRAEQA (p24 161-177), and SILDIKQGKEPFRDY (p24 149-164) contained dominant Gag-speci£c epitopes in 32 out of 37 C-clade infected subjects from South Africa			
p17(77–85)	p17(77–85 LAI)	SLYNTVATL		human(A*0205)	[Brander & Goulder(2001)]
	• C. Brander notes that this epitope can be presented by A*0201 and A*0202				
p17(77–85)	p17()	SLYNTVATL	HIV-1 exposed seronegative	human(A*0214,A*0201)	[Kaul (2000)]
	• 11/16 heavily HIV exposed but persistently seronegative sex-workers in Nairobi had HIV-speci£c CD8 gamma-IFN responses in the cervix – systemic CD8+ T cell responses tended to be to the same epitopes but at generally lower levels than cervical CD8+ T cell responses • Low risk individuals did not have such CD8+ cells • CD8+ epitopes T cell DTVLEDINL (3 individuals), SLYNVATL (4 individuals), LSPRTLNAW (3 individuals) and YPLTFGWCF (4 individuals) were most commonly recognized by the HIV-resistant women • The epitope variants SLYNTVATL and SLFNTVATL were both recognized			
p17(77–85)	Gag(77–85)	SLYNTVATL	HIV A2-polyepitope (polytope) DNA vaccine with vaccinia boost (rVV.HIV.pt)	human(A2)	[Woodberry (1999)]
	• A polyepitope vaccine was generated in a vaccinia construct that contiguously encoded seven epitopes, all presented by HLA A-2 • HHD mice have a transgene of HLA A2 linked to the transmembrane and cytotoxic domains of H-2D^{d} – this transgene is the only MHC molecule expressed in the mice • CTL responses to Gag (77-85) SLYNTVATL, Pol (476-484) ILKEPVHGV, gp120 (120-128) KLTPLCVTL, and Nef (190-198) AFHHVAREL were observed in HIV polytope HHD-vaccinated mice, and these responses were enhanced with vaccinia boost • No CTL immune responses were generated against HLA A2-restricted HIV epitopes Nef 157-166 (PLTFGWCYKL), Pol 346-354 (VIYQYMDDL), and Nef 180-189 (VLEWRFDSRL) • Sixteen HLA A2+ patients were tested for their ability to make CTL responses by peptide restimulation in culture with the epitopes selected for inclusion in the polytope – one individual recognized all seven of these epitopes; 7 patients had CTL cultures able to recognize at least one of the epitopes, and 6 of those 7 recognized more than one epitope, but they were not able to test all peptides for all patients; many patients only had three peptides tested • SLYNTVATL was recognized by 5/16 HLA-A2 patients			

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(77–85)	p17(77–85)	SLYNTVATL	Live recombinant canarypox (CP) virus vaccine containing multiple HIV-1 genes (HIV-1 MN gp120, HIV-1 LAI gp41, HIV-1 LAI Gag, HIV-1 LAI protease)	human(A2)	[Carruth (1999)]
	• CD4+ and CD8+ Gag and Env speci£c CTL responses were detected in only 1/5 vaccinated volunteers, and were not detectable 1 year after vaccination • CTL responses to epitopes SLYNTVATL and TVYYGVPVWK from HIV+ control patients were used as positive controls • The study explored why vaccinees were non-responsive – non-response was not due to inherent defects or differences in the ability of these individuals to process and present antigen • Lack of response to SLYNTVATL led the authors to speculate that the immunodominance of this epitope in natural infections may not be recapitulated by vaccine antigen				
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A2)	[Birk (1998)]
	• Epitope SL9: A study of p17 variation considering known p17 epitopes and individuals with known HLA types revealed that p17 evolution is in¤uenced by immune pressure from CTLs				
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A2)	[Callan (1998)]
	• Epitope SL9: Included as a negative control in a tetramer study of A2-EBV CTL response				
p17(77–85)	p17()	SLYNTVATL	HIV-1 infection	human(A2)	[Wagner (1998)]
	• Epitope SL9: CTL speci£c for HIV epitopes were used to show that the mediators of both the cytolytic (granzyme A was used as the marker) and non-cytolytic (HIV-1 inhibitory chemokines MIP-1 α and RANTES were used as markers) anti-viral responses are localized within the CTL‘s cytotoxic granules				
p17(77–85)	p17(77–85 HXB2)	SLYNTVATL	HIV-1 infection	human(A2)	[Collins (1998)]
	• Epitope SL9: Two CTL clones recognize this epitope, but not the NL4-3 form of the epitope SLYNTIAVL • Nef down-regulates MHC class I molecules, which inhibits CTL killing, and this down-regulation can be partially compensated for by adding excess soluble peptide				
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A2)	[Durali (1998)]
	• Epitope SL9: Cross-clade CTL response was studied by determining the CTL activity in seven patients from Bangui, (6 A subtype, and 1 AG recombinant infections) and one A subtype infection from a person living in France originally from Togo, to different antigens expressed in vaccinia • Pol reactivity: 8/8 had CTL to A subtype, and 7/8 to B subtype, and HIV-2 Pol was not tested • Gag reactivity: 7/8 reacted with A or B subtype gag, 3/8 with HIV-2 Gag • Nef reactivity: 7/8 reacted with A subtype, and 5/8 with B subtype, none with HIV-2 Nef • Env reactivity: 3/8 reacted with A subtype, 1/8 with B subtype, none with HIV-2 Env • Patient B18 had the greatest breadth and diversity of response, and recognized Gag SLYNTVATL and Nef PLTFGWCFKL				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A2)	[Kundu (1998b)]
	• Epitope SL9: Allogeneic dendritic cells (DCs) were obtained from HLA-identical siblings, pulsed with rgp160 MN or A2-restricted HIV-1 epitope peptides, and infused monthly into six HIV-infected patients • 1/6 showed increased env-speci£c CTL and increased lymphoproliferative responses, 2/6 showed increase only in proliferative responses, and 3/6 showed no change – pulsed DCs were well tolerated • SLYNTVATL is a conserved HLA-A2 epitope included in this study – 3/6 patients had this sequence as their HIV direct sequence, one had the form SLYNTVAVL and all four of these had a detectable CTL response – the other two had either the sequence SLFSAVAVL or SLFSAVAAL and no detectable CTL response			
p17(77–85)	p17(77–85 IIIB)	SLYNTVATL	HIV-1 infection	human(A2)	[Sipsas (1997)]
	• Epitope SL9: HIV IIIB proteins were used to de£ne the range of CTL epitopes recognized by 3 lab workers accidentally infected with HIV-1 IIIB • SLYNTVAVL, a variant found in HIV-1 MANC, was also recognized • SLFNTVAVL, a variant found in HIV-1 NY5CG, was also recognized				
p17(77–85)	p17()	SLYNTVATL	HIV-1 infection	human(A2)	[Rowland-Jones (1998a)]
	• Epitope SL9: A CTL response was found in exposed but uninfected prostitutes from Nairobi using previously-de£ned B clade epitopes that tended to be conserved in A and D clades – such cross-reactivity could protect against both A and D and confer protection in Nairobi where both subtypes are circulating • The A subtype consensus is SLfNtvatL • The D subtype consensus is SLyNTvATL				
p17(77–85)	p17()	SLYNTVATL	HIV-1 infection	human(A2)	[Sewell (1997)]
	• Epitope SL9: Naturally occurring variants of this epitope escaped killing and acted as antagonists • The following variants were found in HIV-1 infected patients who mounted a strong response against this epitope: –F——, –F—-V-, –S——, -SF——, –L——, ——I—, ——I-V-, –F–I—, –F–I-V-, –F-A—- • All variants bound to A2 with at least half the af£nity of SLYNTVATL except the triple mutant: –F–I-V- • Antagonism could be observed at low concentrations, abrogating lysis at an antagonist:agonist ratio of 1:10 – the antagonism was observed in one SLYNTVATL-speci£c CTL line but not another				
p17(77–85)	p17(77–85 HXB2)	SLYNTVATL	HIV-1 infection	human(A2)	[Yang (1997b)]
	• Epitope SL9: A chimeric universal T cell receptor was created by linking CD4 or an HIV-speci£c anti-gp41 Ig sequence to the signaling domain of the T cell receptor chain ζ, and transduced into CD8+ cells • The response using universal-receptor-bearing CD8+ cells to lyse infected cells *in vitro* was comparable to the natural occurring responses of CTL-clones from HIV+ individuals in terms of kinetics and ef£ciency • A CTL clone speci£c for this epitope was used for the comparison				

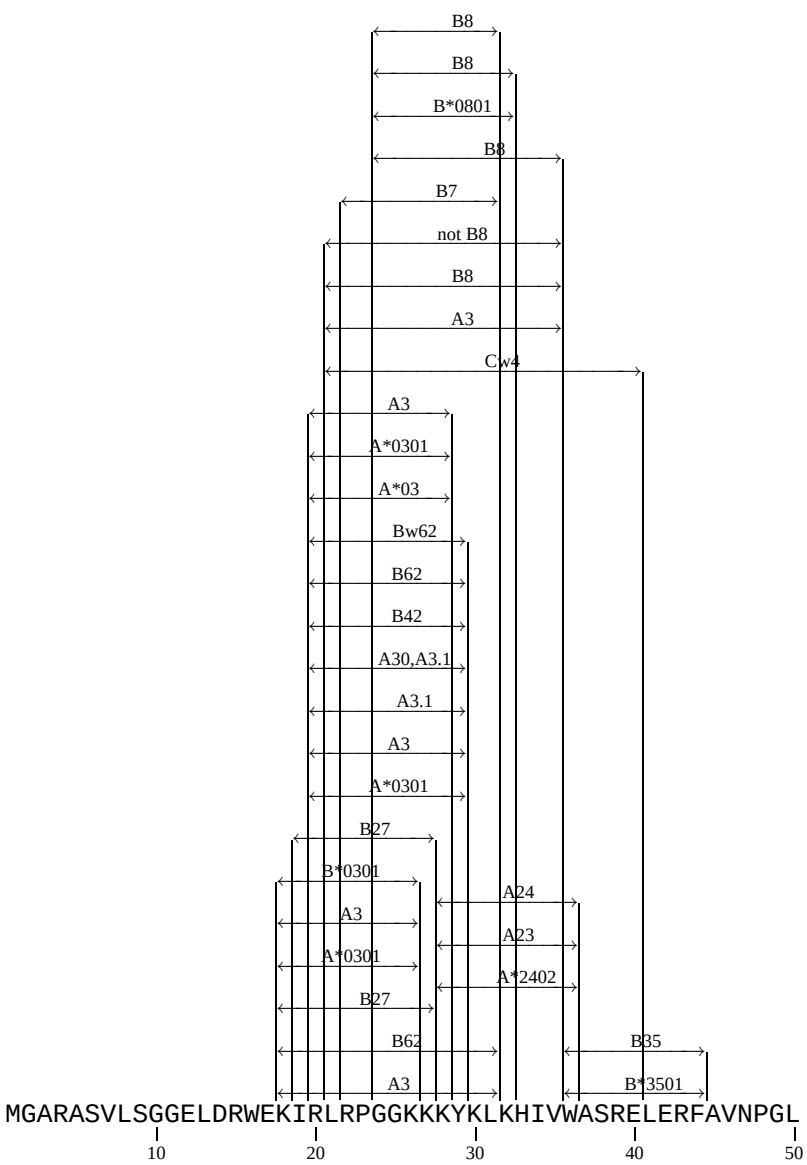
HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(77–85)	p17(77–85)	SLYNTVATL	*in vitro* stimulation	human(A2)	[Stuhler & Schlossman(1997)]
	• Epitope SL9: Keyhole limpit hemocyanin or tetanus toxoid Th epitope co-expression with peptide CTL epitopes on the same APC was required for induction of peptide-speci£c CTL				
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A2)	[Yang (1996)]
	• Epitope SL9: CD4+ cell lines acutely infected with HIV were studied to determine their susceptibility to lysis by CTL • Clones speci£c for RT lysed HIV-1 infected cells at lower levels than Env or Gag speci£c clones • The distinction was thought to be due to lower expression of RT relative to Env and Gag • CTL can lyse infected cells early after infection, possibly prior to viral production				
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A2)	[Yang (1997a)]
	• Epitope SL9: CTL inhibit HIV-1 replication at effector cell concentrations comparable to those found *in vivo* • CTL produced HIV-1-suppressive soluble factors – MIP-1α, MIP-1β, RANTES, after antigen-speci£c activation • CTL suppress HIV replication more ef£ciently in HLA-matched cells				
p17(77–85)	p17(77–85 LAI)	SLYNTVATL	HIV-1 infection	human(A2)	[Parker (1992), Parker (1994)]
	• Epitope SL9: Examined in the context of motifs important for HLA-A2 binding				
p17(77–85)	p17(77–85 LAI)	SLYNTVATL	HIV-1 infection	human(A2)	[McMichael & Walker(1994)]
	• Epitope SL9: Review of HIV CTL epitopes				
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A2)	[Tsomides (1994)]
	• Epitope SL9: CTL clones recognize naturally processed peptide				
p17(77–85)	p17(77–85)	SLYNTVATL	Peptide stimulation *in vitro*	human(A2)	[Stuhler & Schlossman(1997)]
	• Epitope SL9: A three cell-type cluster consisting of APCs, Th, and CTLs is the minimal regulatory unit required for Th cell-dependent induction of CTLs				
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A2)	[Cao (1997)]
	• Epitope SL9: The consensus peptides of B and D clade viruses and some Cs have the sequence SLYNTVATL • The consensus peptide of A, and some C strains have SLFNTVATL, a form that is cross-reactive				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(77–85)	Gag(77–85)	SLYNTVATL	HIV-1 infection	human(A2)	[Dyer (1999)]
• Epitope SL9: CTL speci£c responses were measured over a 1.3 to 1.5 year period in members of the Sydney Blood Bank Cohort (SBBC) who had been infected with a natural attenuated strain of HIV-1 which was Nef-defective • Some of these patients had prolonged high levels of CTL effector and memory cells despite low viral load					
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(A2)	[Harrer (1998)]
• Epitope SL9: Two overlapping epitopes were recognized in a long-term survivor, restricted by two different HLA molecules, HLA-A11(TLYCVHQR) and -A2 (SLYNTVATL) • Viral sequence substitutions were present in this individual which did not affect viral replication and did not alter CTL-recognition of the A2 epitope, but reduced recognition of the A11 epitope, indicative of immune escape					
p17(77–85)	p17()	SLYNTVATL	HIV-1 exposure	human(A2, A*0202)	[Rowland-Jones (1998b)]
• Epitope SL9: HIV-speci£c CTL were found in exposed seronegative prostitutes from Nairobi – these CTL may confer protection • Seroprevalence in this cohort is 90-95% and their HIV-1 exposure is among the highest in the world • Most isolated HIV strains are clade A in Nairobi, although clades C and D are also found – B clade epitopes are often cross-reactive, however stronger responses are frequently observed using A or D clade versions of epitopes • This epitope is conserved among B and D clade viruses • The Clade A version of the epitope, SLFNTVATL, was preferentially recognized by CTL • This epitope was recognized by two different exposed seronegative prostitutes					
p17(77–85)	p17()	SLYNTVATL	HIV-1 infection	human(B*0201)	[Wilson (2000)]
• Three individuals with highly focused HIV-speci£c CTL responses were studied during acute infection using tetramers – high frequencies of HIV-1-speci£c CD8+ T cells were found prior to seroconversion, and there was a close temporal relationship between the number of circulating HIV-speci£c T cells and viral load was also found • All three patients were B*2705, with HLA alleles: A1, A30/31, B*2705, B35; A1, A*0301, B7, B2705; and A*0201, A*0301, B2705, B39 • ELISPOT was used to test a panel of CTL epitopes that had been de£ned earlier and were appropriate for the HLA haplotypes of the study subjects – 3/3 subjects showed a dominant response to the B*2705 epitope KRWIILGGLNK • The subject with A*0201 had a moderatly strong response to SLYNTVATL • Weak responses were observed to A*301-RLRPGGKKK, A*301-QVPLRPMTYK, and B7-TPGPGVRYPL in the subject who was HLA A1, A*0301, B7, B*2705 • No acute response was detected to the following epitopes: A*201-ILKEPVHGV, A*301-KIRLRPGGK, A*301-AIFQSSMTK, A*301-TVYYGVPVWK, B35-EPIVGAETF, B35-HPDIVIYQY, B35-PPIPVGEIY, B35-NSSKVSQNY, B35-VPLRPMTY, B35-DPNPQEVVL			

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(77–85)	p17(77–85)	SLYNTVATL	HIV-1 infection	human(B62)	[Goulder (1997a)]

- Epitope SL9: This paper is a review of CTL and immune evasion, but it presents a study of a shift from an HLA-A*0201 response to SLYNTVATL, to a B62 response to GLNKIVRMY
- As long as a strong CTL response to SLYNTVATL was evident, the epitope variants SLFNTVATL or SLYNTIATL dominated the viral population – eventually the CTL response to the index peptide became undetectable, the CTL response shifted to a focus on GLNKIVRMY, and the index peptide SLYNTVATL once again established itself as the dominant form

p17 CTL Map


B8
B8
B*0801
B8
B7
not B8
B8
A3
Cw4
A3
A*0301
A*03
Bw62
B62
B42
A30,A3.1
A3.1
A3
A*0301
B27
B*0301
A3
A*0301
B27
B62
A3
A24
A23
A*2402
B35
B*3501
MGARASVLSGGELDRWEKIRLRPGGKKKYKLKHIVWASRELERFAVNPGL
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20
30
40
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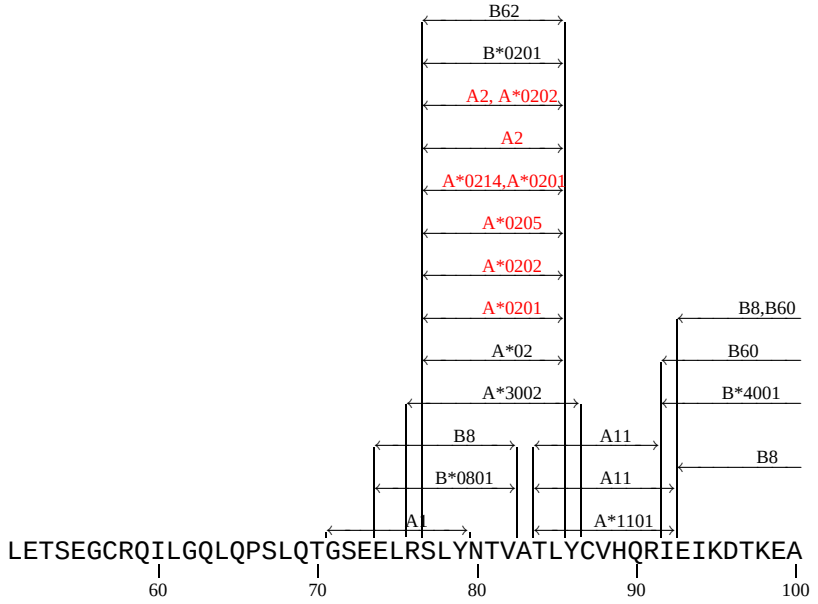
B62
B*0201
A2, A*0202
A2
A*0214,A*0201
A*0205
A*0202
A*0201
A*02
A*3002
B8
B*0801
A1
A11
A11
A*1101
B8,B60
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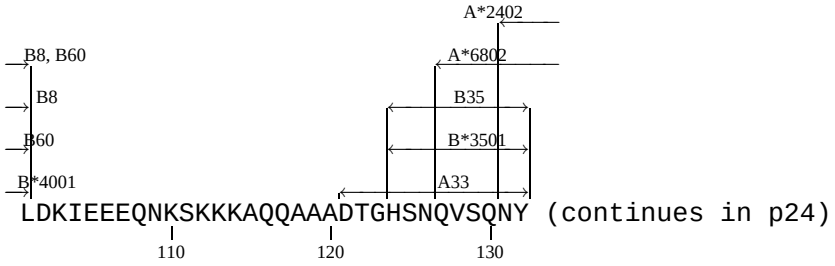
70
80
90
100


A*2402
A*6802
B35
B*3501
A33
B8, B60
B8
B60
B*4001
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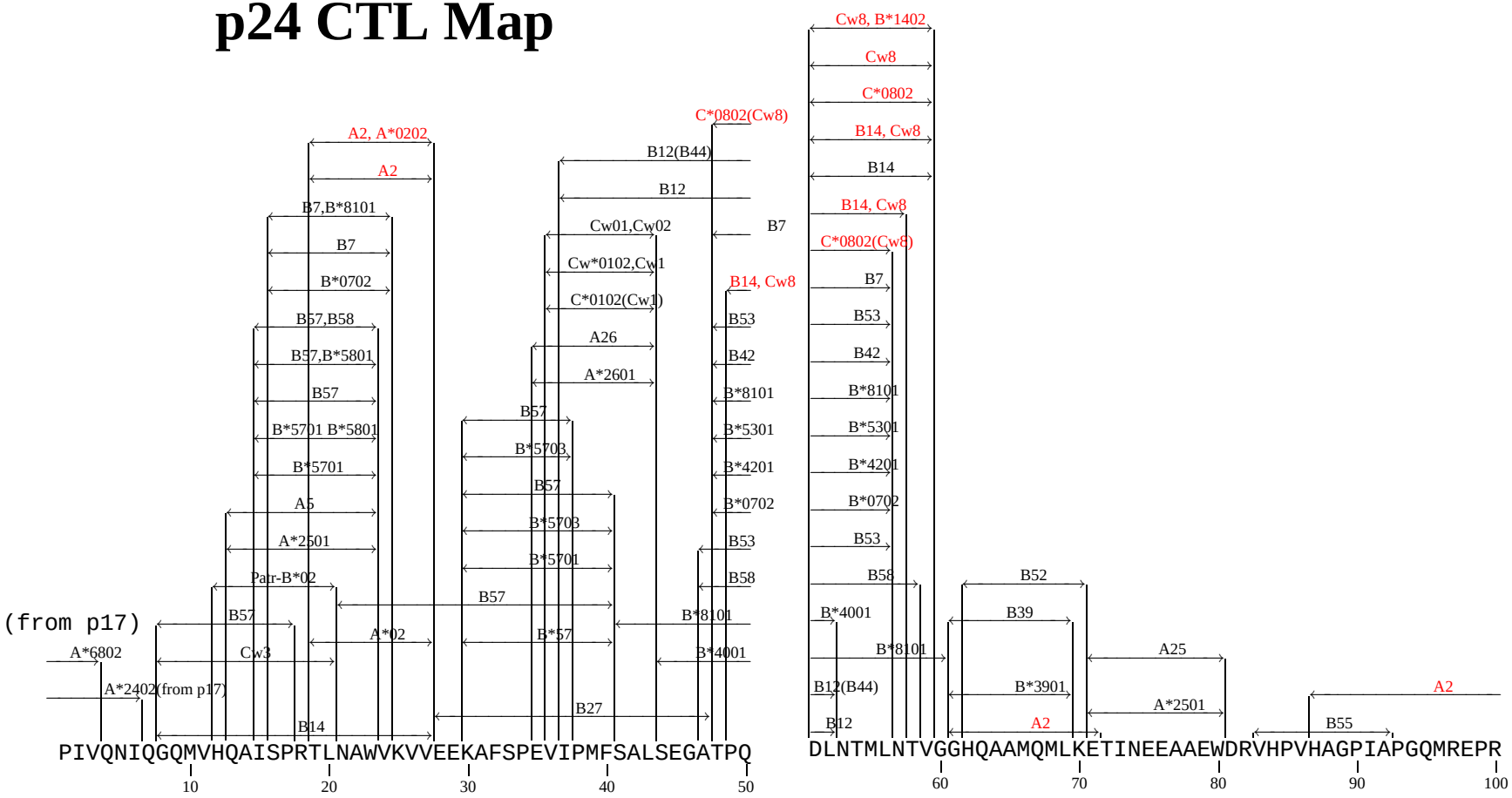
110
120
130

p24 CTL Map


(from p17)
A*6802
A*2402(from p17)
A2, A*0202
A2
B7,B*8101
B7
B*0702
B57,B58
B57,B*5801
B57
B*5701 B*5801
B*5701
A5
A*2501
Pair-B*02
B57
A*02
Cw3
B14
C*0802(Cw8)
B12(B44)
B12
Cw01,Cw02
Cw*0102,Cw1
C*0102(Cw1)
A26
A*2601
B57
B*5703
B57
B*5703
B*5701
B57
B*8101
B*57
B7
B14, Cw8
B53
B42
B*8101
B*5301
B*4201
B*0702
B53
B58
B*8101
B*4001
B14
B27
PIVQNIQGQMVHQAISPRTLNAWVKVVEEKAFSPEVIPMFSALSEGATPQ
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20
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40
50
Cw8, B*1402
Cw8
C*0802
B14, Cw8
B14
B14, Cw8
C*0802(Cw8)
B7
B53
B42
B*8101
B*5301
B*4201
B*0702
B53
B58
B*4001
B*8101
B12(B44)
B12
B52
B39
A25
B*3901
A2
A*2501
A2
B55
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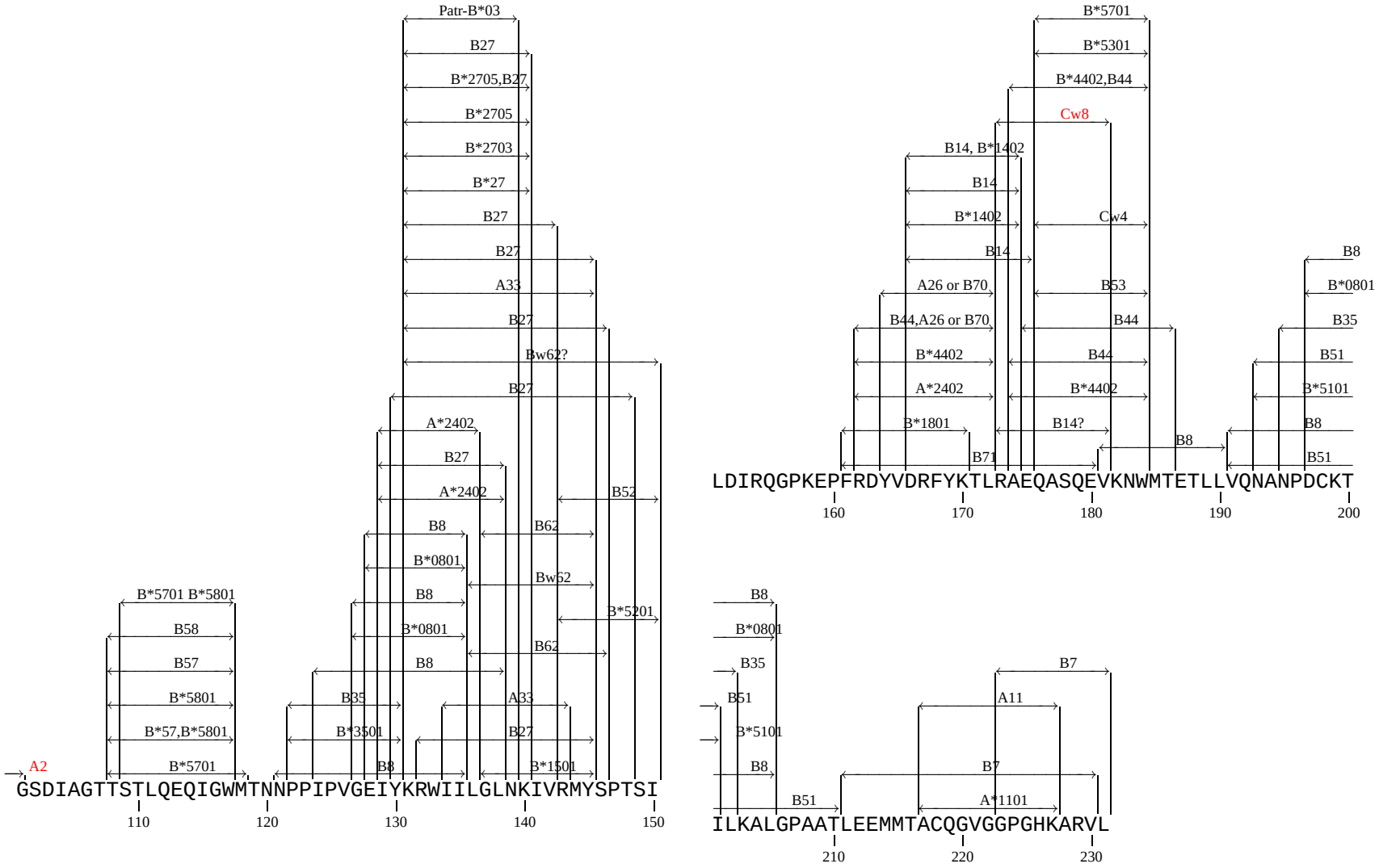
Patr-B*03
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B*2705,B27
B*2705
B*2703
B*27
B27
B27
A33
B27
Bw62?
B27
A*2402
B27
A*2402
B52
B8
B62
B*0801
Bw62
B*5701 B*5801
B8
B*5201
B58
B*0801
B62
B57
B8
B*5801
B35
A33
B*57,B*5801
B*3501
B27
A2
B*5701
B8
B*1501
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110
120
130
140
150
B*5701
B*5301
B*4402,B44
Cw8
B14, B*1402
B14
B*1402
Cw4
B14
B8
A26 or B70
B53
B*0801
B44,A26 or B70
B44
B35
B*4402
B44
B51
A*2402
B*4402
B*5101
B*1801
B14?
B8
B8
B71
B51
LDIRQGPKEPFRDYVDRFYKTLRAEQASQEVKNWMTETLLVQNANPDCKT
160
170
180
190
200
B8
B*0801
B35
B7
B51
A11
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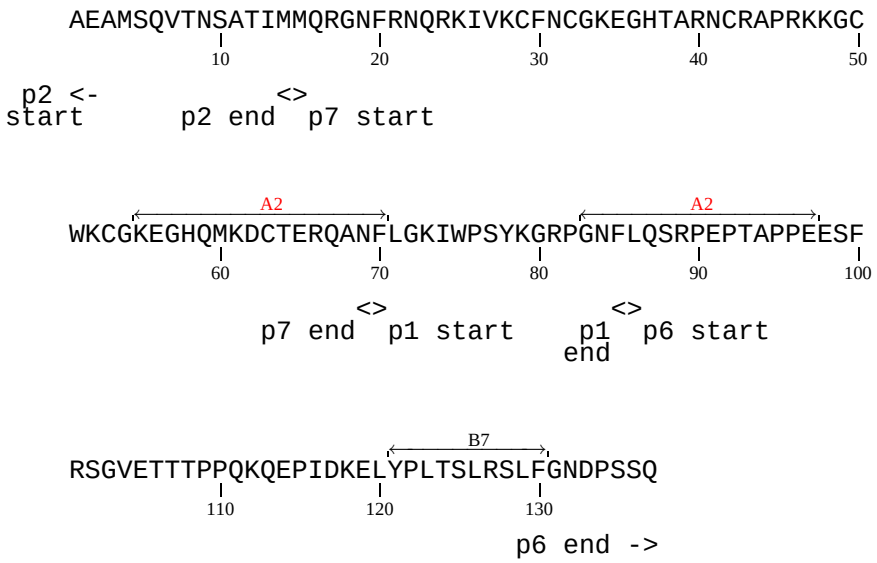
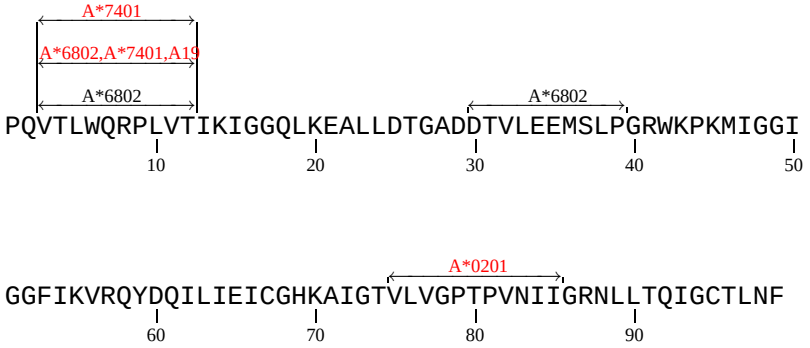
220
230

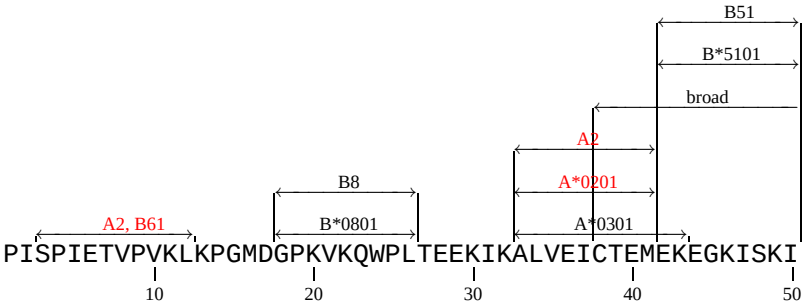
p2p7p1p6 CTL Map



Protease CTL Map



RT CTL Map


B51
B*5101
broad
A2
B8
A*0201
A2, B61
B*0801
A*0301
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30
40
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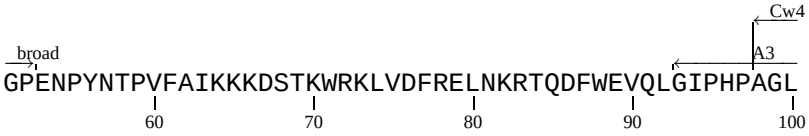
Cw4
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A3
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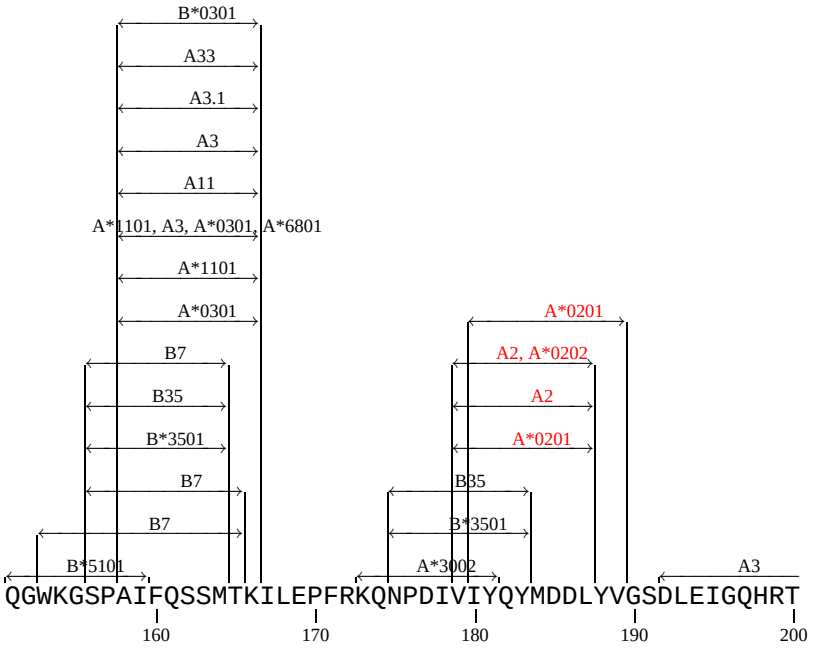
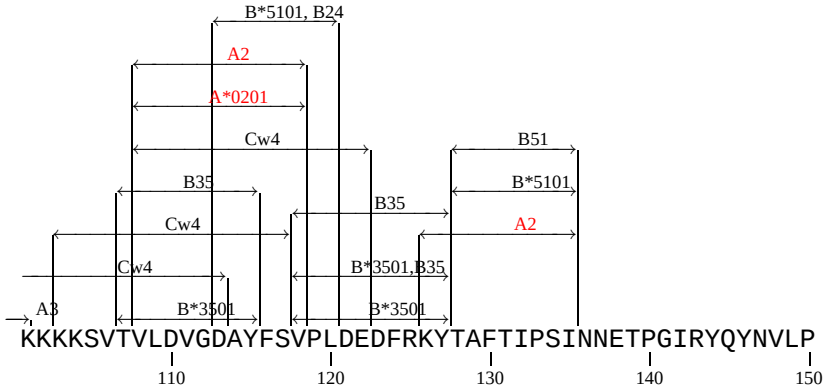
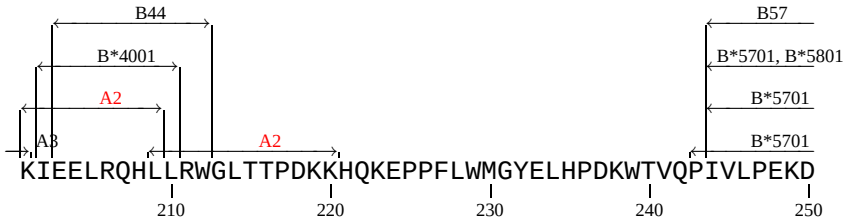
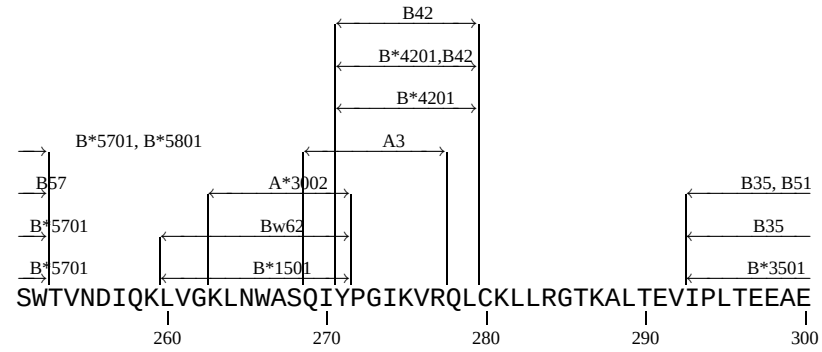
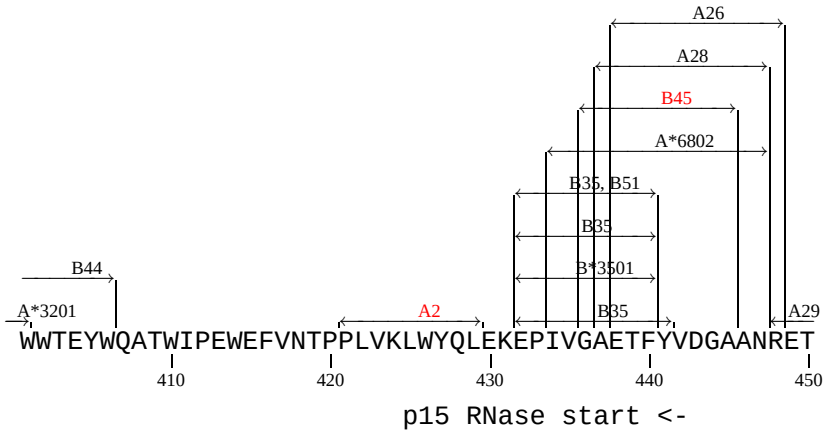

B*0301
A33
A3.1
A3
A11
A*1101, A3, A*0301, A*6801
A*1101
A*0301
B7
B35
B*3501
B7
B7
B*5101
A*0201
A2, A*0202
A2
A*0201
B35
B*3501
A*3002
A3
QGWKGSPAIFQSSMTKILEPFRKQNPDIVIYQYMDDLYVGSDLEIGQHRT
160
170
180
190
200

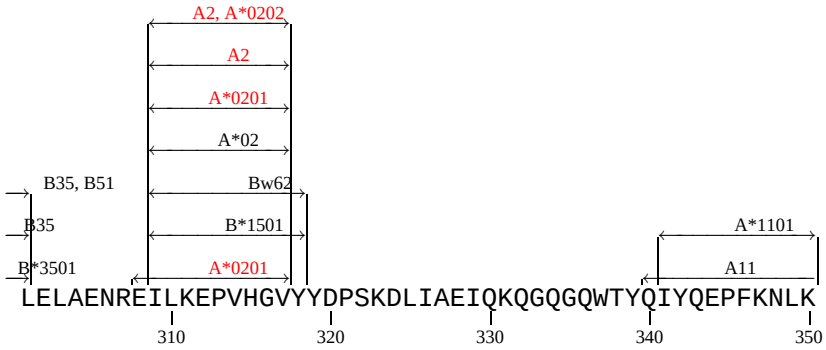

B*5101, B24
A2
A*0201
Cw4
B51
B35
B*5101
B35
Cw4
A2
Cw4
B*3501,B35
A3
B*3501
B*3501
KKKKSVTVLDVGDAYFSVPLDEDFRKYTAFTIPSINNETPGIRYQYNVLP
110
120
130
140
150

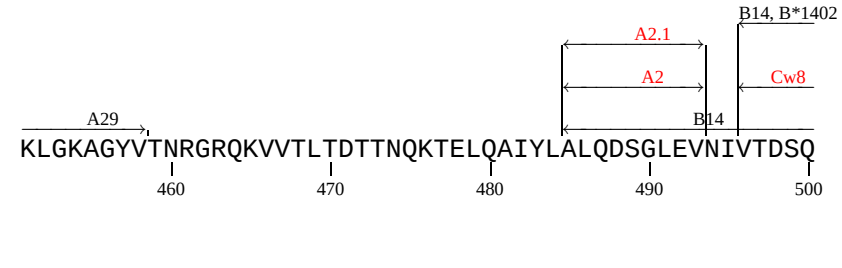

B44
B57
B*4001
B*5701, B*5801
A2
B*5701
A3
A2
B*5701
KIEELRQHLLRWGLTTPDKKHQKEPPFLWMGYELHPDKWTVQPIVLPEKD
210
220
230
240
250

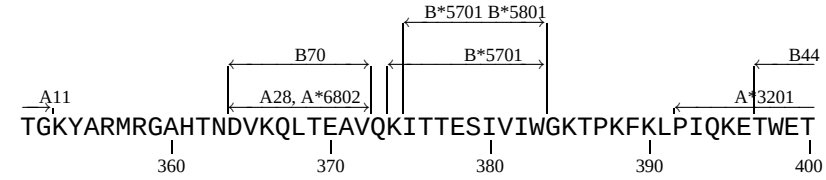


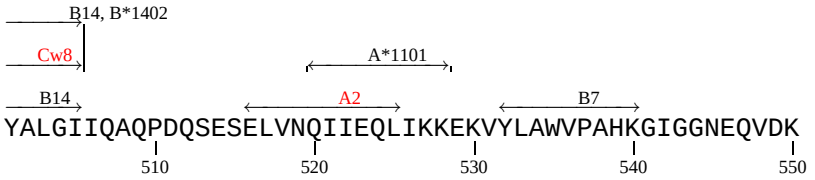


p15 RNase start <-





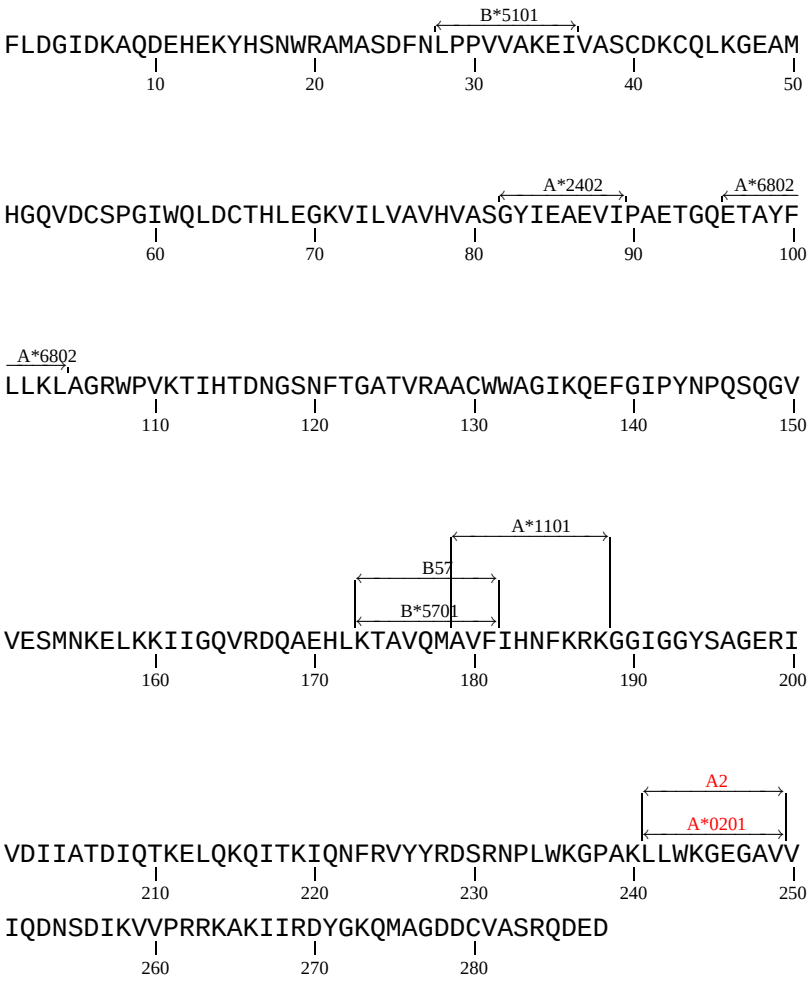




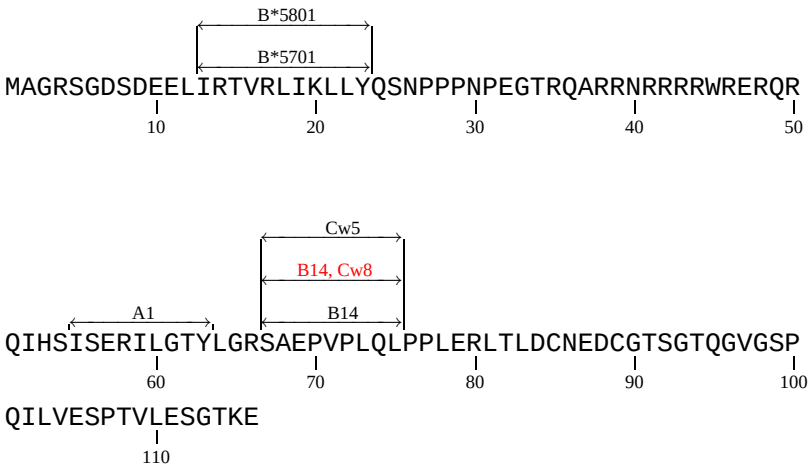
LVSAGIRKVL

-> p15 RNase end

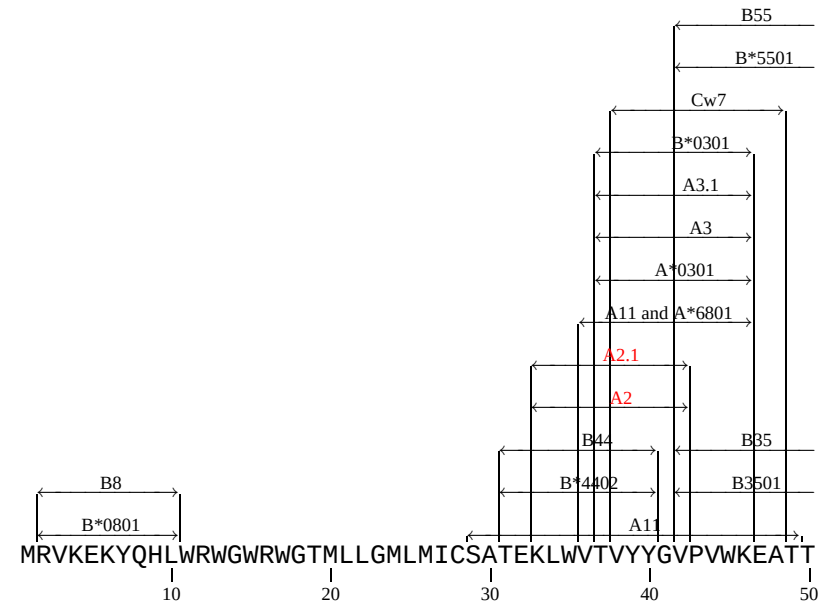
Integrase CTL Map



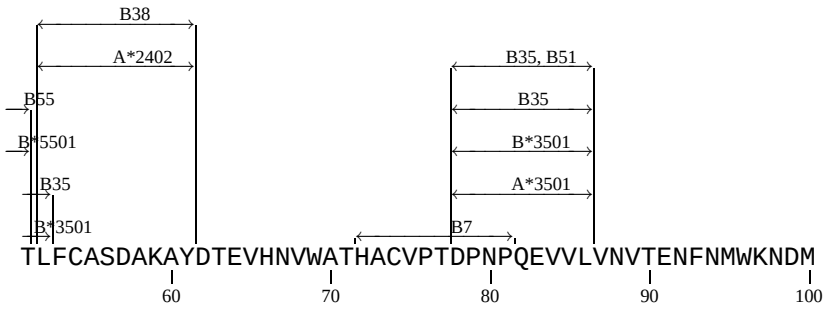
Rev CTL Map

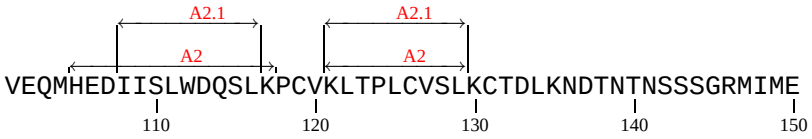


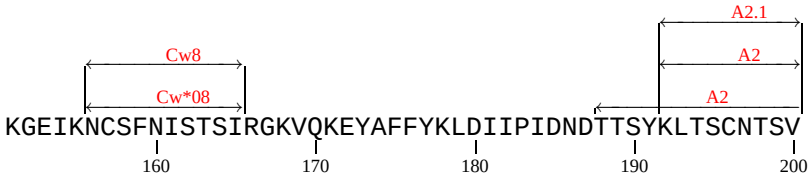
gp160 CTL Map

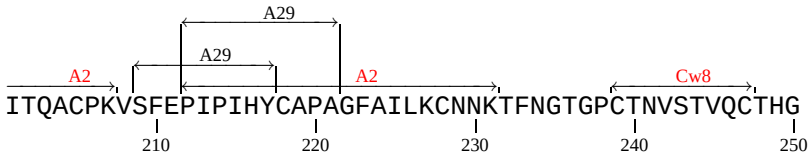


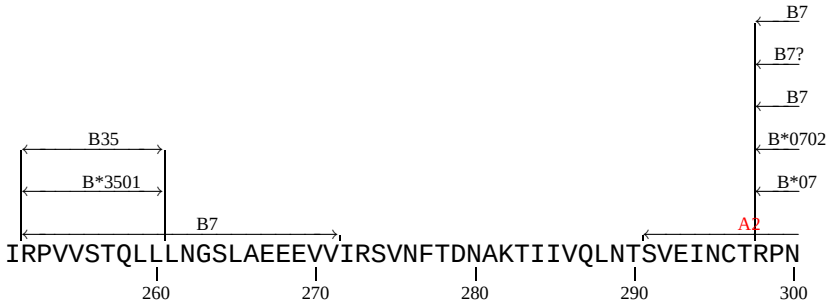
<- gp120 start

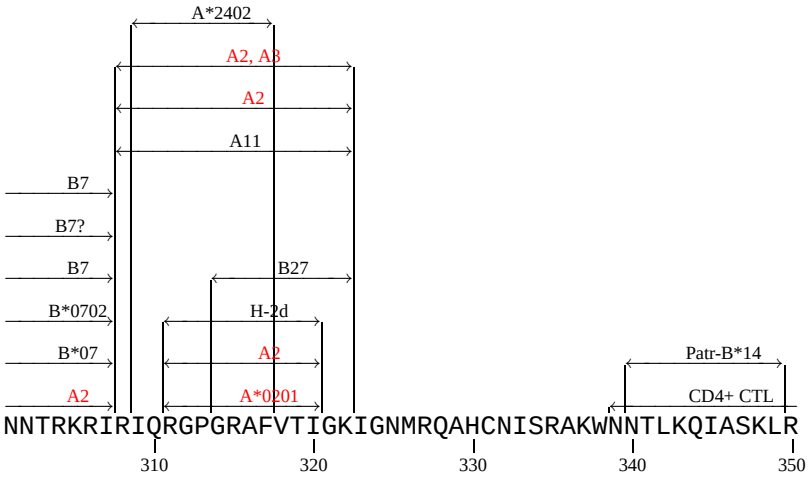


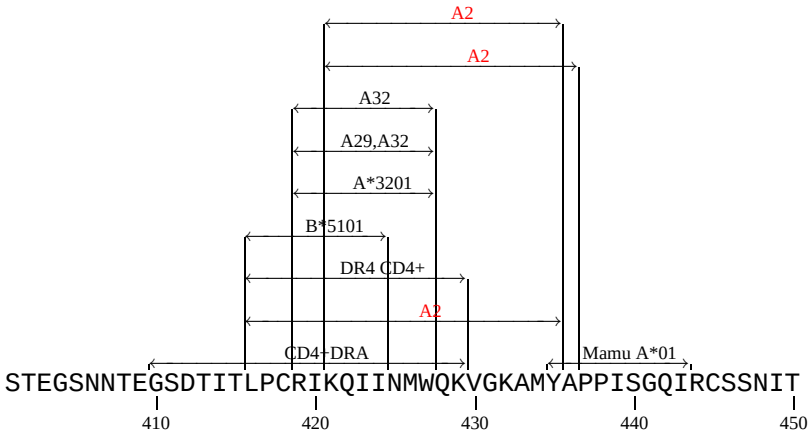


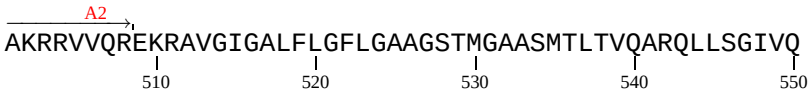




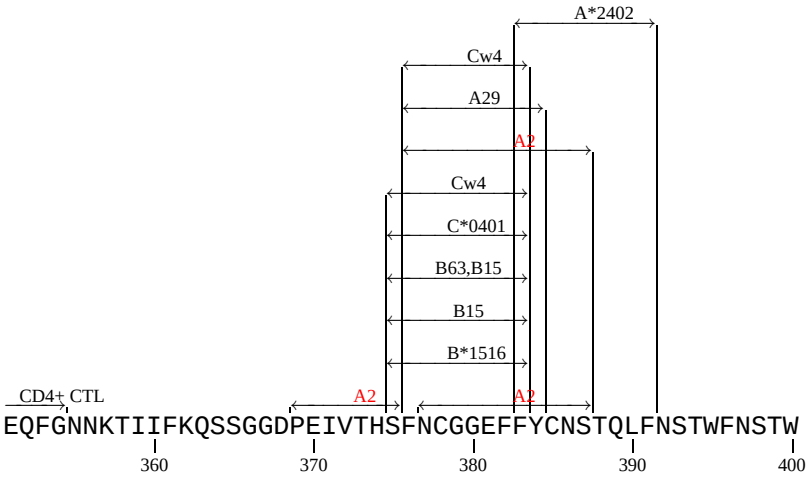


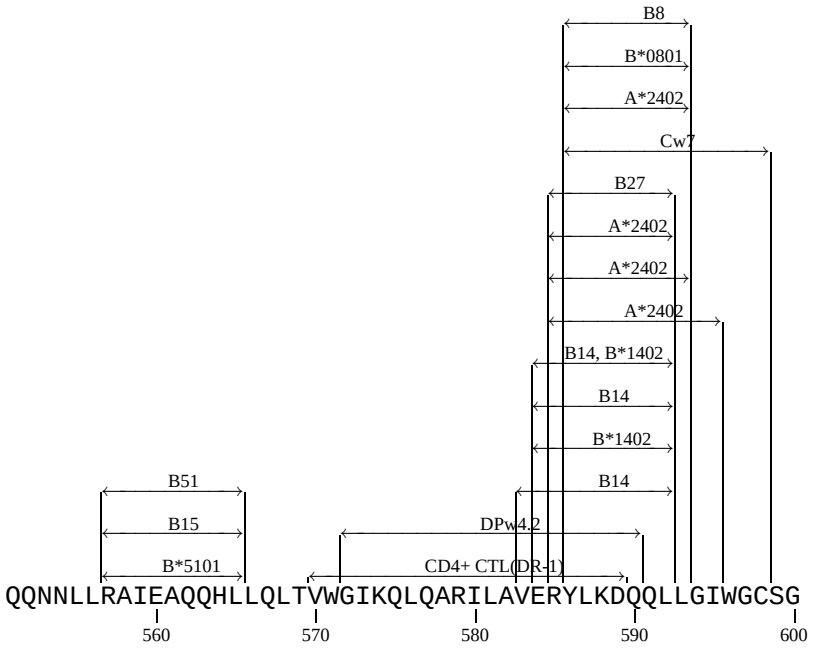


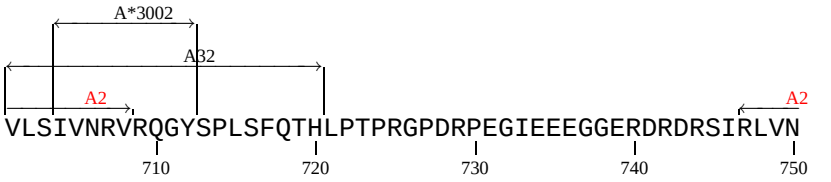


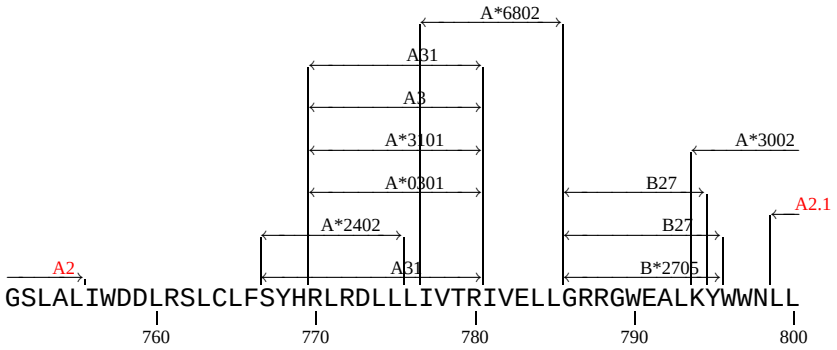


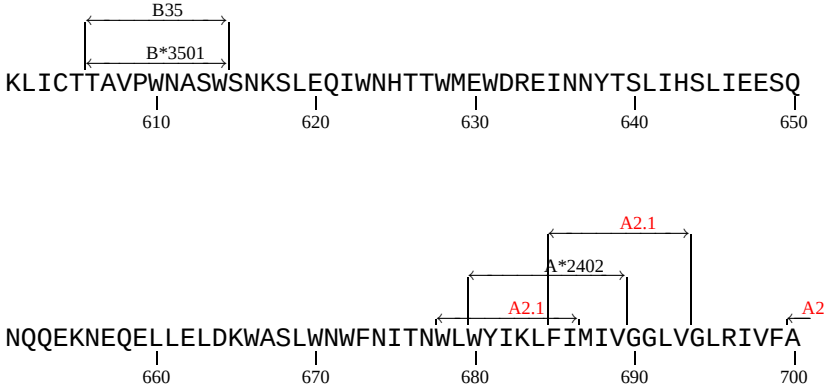
gp120 end <> gp41 start



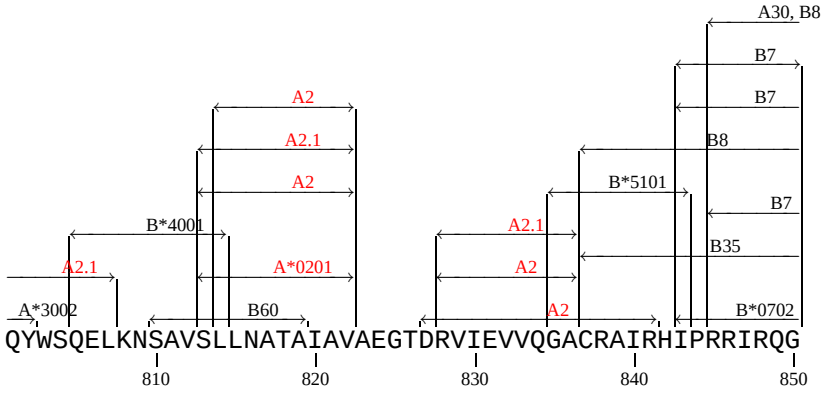

B8
B*0801
A*2402
Cw7
B27
A*2402
A*2402
A*2402
B14, B*1402
B14
B*1402
B14
B51
B15
B*5101
DPw4.2
CD4+ CTL(DR-1)
QQNNLLRAIEAQQHLLQLTVWGIKQLQARILAVERYLKDQQLLGIWGCSG
560
570
580
590
600

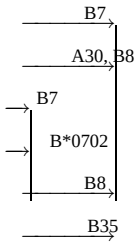

A*3002
A32
A2
A2
VLSIVNRVRQGYSPLSFQTHLPTPRGPDRPEGIEEEGGERDRDRSIRLVN
710
720
730
740
750


A*6802
A31
A3
A*3101
A*0301
A*2402
A31
A*3002
B27
B27
B*2705
A2.1
A2
GSLALIWDDLRSLCLFSYHRLRDLLLIVTRIVELLGRRGWEALKYWWNLL
760
770
780
790
800


B35
B*3501
KLICTTAVPWNASWSNKSLEQIWNHTTWMEWDREINNYTSLIHSLIEESQ
610
620
630
640
650

A2.1
A*2402
A2.1
A2
NQQEKNEQELLELDKWASLWNWFNITNWLWYIKLFIMIVGGLVGLRIVFA
660
670
680
690
700

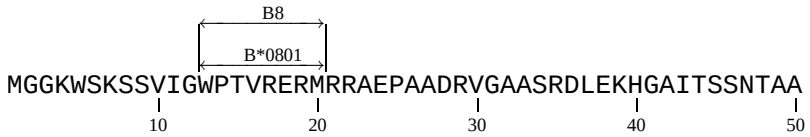

A30, B8
B7
A2
B7
A2.1
B8
A2
B*5101
B*4001
A2.1
B7
A2.1
B35
A*3002
A*0201
A2
B60
A2
B*0702
QYWSQELKNSAVSLLNATAIAVAEGTDRVIEVVQGACRAIRHIPRRIRQG
810
820
830
840
850

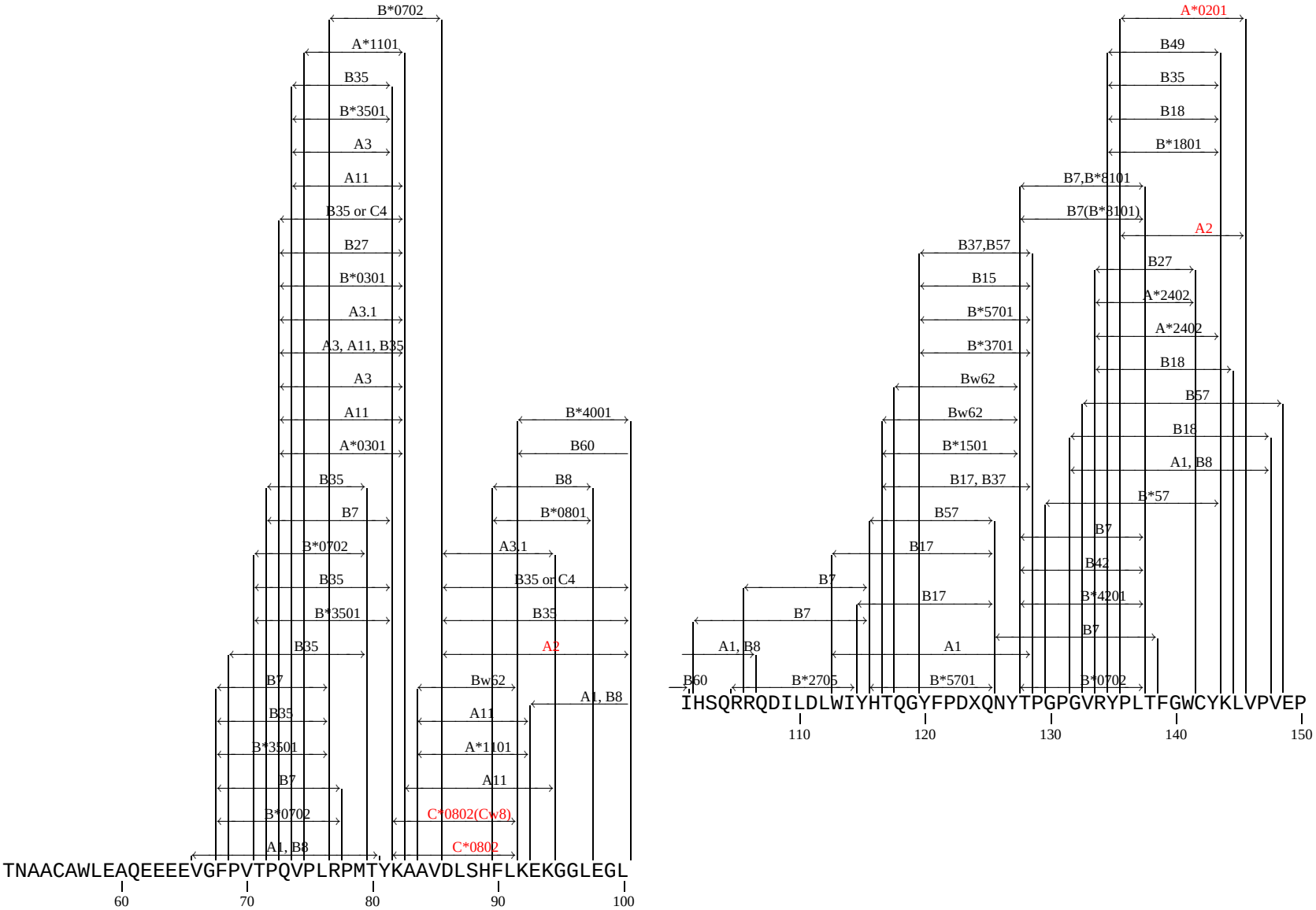


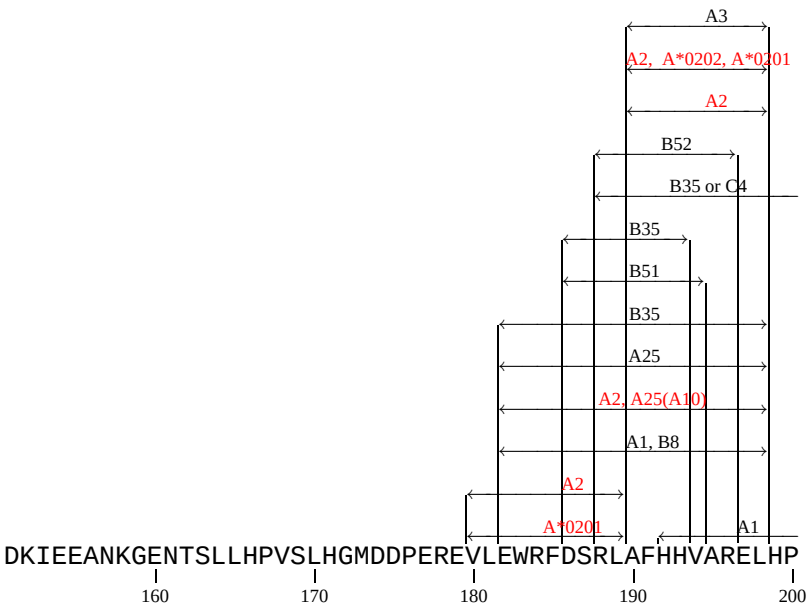
LERILL

-> gp41 end

Nef CTL Map




B*0702
A*1101
B35
B*3501
A3
A11
B35 or C4
B27
B*0301
A3.1
A3, A11, B35
A3
A11
A*0301
B35
B7
B*0702
B35
B*3501
B35
B7
A1, B8
B*4001
B60
B8
B*0801
A3.1
B35 or C4
B35
A2
Bw62
A11
A*1101
A11
C*0802(Cw8)
C*0802
TNAACAWLEAQEEEEVGFPVTPQVPLRPMTYKAAVDLSHFLKEKGGLEGL
60
70
80
90
100
A*0201
B49
B35
B18
B*1801
B7,B*8101
B7(B*3101)
A2
B37,B57
B27
B15
A*2402
B*5701
A*2402
B*3701
B18
Bw62
B57
Bw62
B18
B*1501
A1, B8
B17, B37
B*57
B57
B7
B17
B42
B7
B17
B*4201
B7
B7
A1, B8
A1
B60
B*2705
B*5701
B*0702
IHSQRRQDILDLWIYHTQGYFPDXQNYTPGPGVRYPLTFGWCYKLVPVEP
110
120
130
140
150



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responses in two human histocompatibility leukocyte antigens (HLA)-identical siblings with HLA-A*0201 are in¤uenced by epitope mutation. *J Exp Med* **8**:1423–33, 1997b. (Medline: 97272078) Notes: Primary human immunode£ciency virus (HIV) infection is controlled principally by HIV-speci£c cytotoxic T lymphocytes (CTL) to a steady- state level of virus load, which strongly in¤uences the ultimate rate of progression to disease. Epitope selection by CTL may be an important determinant of the degree of immune control over the virus. This report describes the CTL responses of two HLA-identical hemophiliac brothers who were exposed to identical batches of Factor VIII and became seropositive within 10 wk of one another. Both have HLA-A*0201. The CTL responses of the two siblings were very dissimilar, one donor making strong responses to two epitopes within p17 Gag (HLA-A*0201-restricted SLYNTVATL and HLA-A3-restricted RLRPGGKKK). The sibling responded to neither epitope, but made strong responses to two epitopes presented by HLA-B7. This was not the result of differences in presentation of the epitopes. However, mutations in both immunodominant epitopes of the p17 Gag responder were seen in proviral sequences of the nonresponder. We then documented the CTL responses to two HLA-A*0201-restricted epitopes, in Gag (SLYNTVATL) and Pol (ILKEPVHGV) in 22 other HIV-infected donors with HLA-A*0201. The majority (71%) generated responses to the Gag epitope. In the 29% of donors failing to respond to the Gag epitope in standard assays, there was evidence of low frequency memory CTL responses using peptide stimulation of PBMC, and most of these donors also showed mutations in or around the Gag epitope.

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identi£cation of group-speci£c CTL responses and precluded assessment of the extent of type-speci£c CTL responses directed against HIV-1. Using cells expressing viral proteins from the HIV-1 IIIB strain, we performed a detailed characterization of HIV-1-speci£c CTL response in three laboratory workers accidentally infected with HIV-1 IIIB. Eight of the epitopes identi£ed were group speci£c, lying in relatively conserved regions of Gag, reverse transcriptase, and envelope. Three type-speci£c epitopes were identi£ed, two of them in highly variable regions of envelope. In longitudinal studies in one subject, seven different epitopes and £ve different restricting HLA class I alleles were identi£ed, with a progressive increase in the number of CTL epitopes recognized by this subject over time. Our data demonstrate that type-speci£c CTL responses make up a signi£cant proportion of the host cellular immune response against HIV-1 and that a broadening of epitope speci£city may occur.

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